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REMARKS ON THE FIELD OF VISION IN CERTAIN CASES OF "NEGLECTED EYES."

BY G. E. DE SCHWEINITZ, M. D.,
OF PHILADELPHIA.[1]

THE following cases illustrate some of the visual-field phenomena in that type of monocular amblyopia which is common with convergent and divergent strabismus. Although we may be familiar with the changes here depicted, the field of vision of "neglected eyes" is not always carefully investigated, and I believe that greater attention to this method of examination would add to our knowledge of these amblyopias.

[1] Read before the Ophthalmic Section of the College of Physicians of Philadelphia, March, 1895.

To introduce the subject I quote a paragraph written by Dr. Henry D. Noyes:

"Monocular amblyopia is very common in strabismus convergens and not infrequent in strabismus divergens. This may or may not be associated with high degrees of hyperopia or with astigmatism, possibly irregular. We meet with it where the degree of ametropia differs little from that of the eye with good vision, and in a very large proportion, perhaps in the majority of cases, no lesion can be found with the ophthalmoscope. On this point it is important to bestow careful attention. No small number of cases exhibit what are evidently congenital abnormalities in the papilla. In my records are such conditions as follows: An extraordinary amount of pigment deposit along the border; the presence of connective tissue on edge of nerve and running along the vessels (not to be confounded with opaque nerve fibers); a dull or slaty-colored and opaque disc with hazy edges; extreme hyperemia, both of capillaries and veins; the nerve swollen as in papillitis, a dark gray or slaty spot upon the disc and the rest of the surface an opaque white; coloboma of the sheath of the nerve or a very deep and irregular excavation which was so interpreted. Besides, one must carefully scrutinize the macula and it must be done with dilated pupil. Not rarely will one find minute specks—white, yellow or glistening—clustered here, which indicate lesion either of the choroid or retina. There may be one or more marked pigment specks which will denote a previous inflammatory lesion. A notable number of cases, and the majority, will not reveal any visible lesion. In the examination of the visual field we are often prevented from attaining exact knowledge by the extreme youth of the subjects. When, however, they are sufficiently intelligent, we frequently find that the amblyopia is central, and a defined scotoma for red may be sometimes mapped out provided a small card 5 *mm.* square and dim light can be employed. The scotoma may be very small and will be better discovered on a dark plane surface than by the perimeter. Sometimes a patient will say that over a small space, not the blind spot of Mariotte, a small candle flame is not perceived. This means a small absolute scotoma. In one case I found nasal (medial) amblyopia with the line of demarcation vertical. It was not difficult, to show the decided difference in perceptive power of the respective halves of the retina—this might be called hemiamblyopia."

For the purpose of study I have gathered the cases into three groups, as follows:

GROUP I. CASES IN WHICH THE VISUAL FIELD FOR FORM AND COLORS IS NORMAL, OR PRACTICALLY NORMAL, AND IN WHICH THE ACCURACY OF COLOR PERCEPTION AT THE MACULA, OR BETWEEN IT AND THE FIXING POINT, IS UNAFFECTED.

Two examples will suffice:

Case I. Mrs. P., 39 years of age; denies strabismus, but has never seen well with the left eye. As ordinarily observed, there is no abnormal convergence, but the eyes wander in under cover and there is slight convergence of the left one on fixation at 30 *cm.*, or if tested by means of the corneal reflex of the ophthalmoscope. The right eye is hypermetropic and slightly astigmatic and

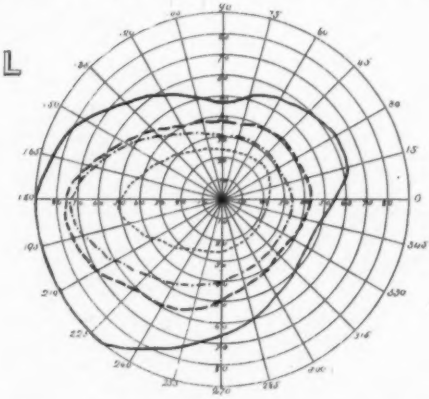

FIG. 1.

Diagram of the field of vision of Case 1, Group 1. Normal in all respects.

possesses full acuity of vision. The vision of the left eye equals counting fingers at 1 foot, the refraction is + 3.50 S. ⊃ + .75 C., axis V., the corneal astigmatism with the Javal ophthalmometer being 1.50 diopters according to the rule, axis 90°.

There is no ophthalmoscopic abnormality in the right eye; in the left the disc is a vertical oval, of good color, contains a small physiological cup, and is bounded by a pigment crescent on the temporal side; the vessels are of normal size.

After wearing the correction for nearly a year, the patient returned with the vision of the right eye $\frac{6}{5}$, of the left eye $\frac{6}{30}$. She had "amused herself by practising," as she expressed it, with her left eye.

The field of vision[2] (Fig. 1) is practically normal in all respects, and there is nowhere an area of diminished color perception nor a scotoma, save the natural blind spot, which is promptly acknowledged.

Case II. Mrs. J., 26 years of age, had convergent strabismus of the left eye in childhood, but "outgrew it." There is now a slight convergence, about 1 *mm.* Vision equals $\frac{6}{40}$. The right eye, save for a slight hypermetropic astigmatism, is entirely normal, and possesses full visual acuity.

In the left eye the optic disc is a vertical oval, rather hyperemic, a slight crescent bounding its temporal border. The ophthalmometer reveals a corneal astigmatism of 1 diopter according to the rule, with its axis vertical, and the refraction of the eye is

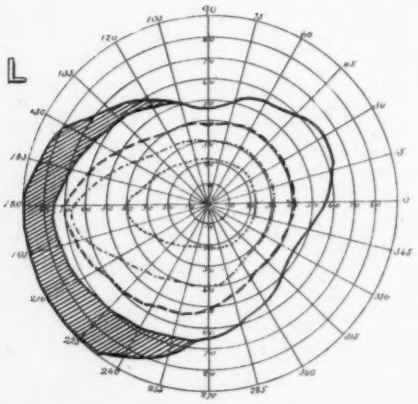

FIG. 2.

Diagram of the field of vision of Case 2, Group 1. Color field practically normal; slight contraction on the temporal side of the form field.

+ 1 S. ⌒ + 0.50 C., axis V. With this glass vision equals $\frac{6}{30}$. Eighteen months later the vision of this eye was about the same, perhaps somewhat better, $\frac{6}{22}$ being recorded.

The field of vision (Fig. 2), save for slight contraction on the temporal side, is practically normal and the color sense of normal standard.

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[2] All the diagrams of the visual fields are constructed as follows: White, ———; blue, — — — —; red, — . — . — . —.; green, ... Scotomas are represented by dotted areas; peripheral contractions by shading with parallel lines.

GROUP II. CASES CHARACTERIZED (a) BY CONTRACTION OF ONE OR MORE OF THE COLOR FIELDS, THE FORM FIELD REMAINING NORMAL, AND (b) BY IRREGULAR CONTRACTION OF BOTH FORM AND COLOR FIELDS, SOMETIMES ASSOCIATED WITH REVERSAL OF THE RED AND BLUE LINES.

Case I. S. Y., a man 22 years of age, with a vague history of inflammation of his eyes in childhood, of which there are no traces, presented a monocular strabismus of the left eye, the angle of squint being 28°. Vision in this eye equaled counting fingers at 1 *m*. There is no corneal astigmatism, but a hypermetropia of 4 D. at the macula. The disc is a vertical oval, of good color; the

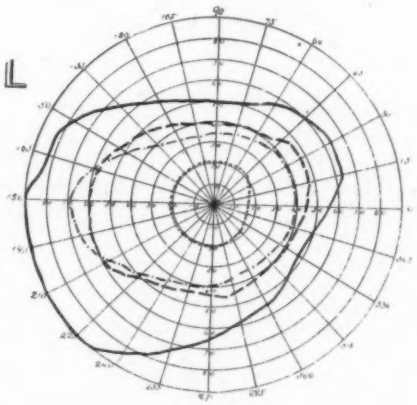

FIG. 3.

Diagram of the field of vision of Case 1, Group 2, exhibiting partial reversal of the red and blue lines, with marked restriction of the green field.

vessels are distorted and pass to the nasal side. After glasses were ordered, and tenotomy of the internus and advancement of the externus had been performed, there was restoration of parallelism of the visual axes, but no improvement in vision. The right eye is slighly hypermetropic, but normal in all other respects.

The dimensions of the form field are normal. There is slight contraction of the blue field and partial reversal of the red and blue lines, with marked restriction of the green field, but no scotoma and no diminution of central color perception. (Fig. 3.)

Case II. Annie J., 25 years of age, has marked divergent strabismus of the left eye, the angle of squint being 34° and the

myopia 16 diopters. The optic disc, surrounded by a posterior staphyloma, is distinctly gray in its deeper layers. The vessels are normal in size and distribution; V. equals counting fingers at 1 foot. The right eye, save for slight myopia, is normal.

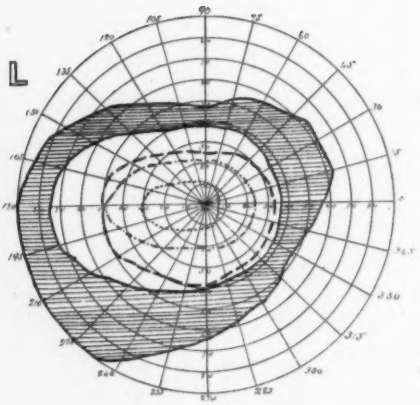

FIG. 4.

Diagram of the field of vision of Case 2, Group 2, showing marked contraction of form and color fields, with practically a half defect in the contracted green field.

The visual field (Fig. 4) shows marked contraction of form and color fields, the restriction being relatively greater for green, which is particularly defective upon the nasal side, practically a half defect, or hemiachromatopsia for this color.

Case III. C. E., a boy 16 years of age, had whooping-cough in childhood, and since then divergent strabismus of the right eye in which the vision amounts to counting fingers at 2 feet. The exact refraction of the eye is — 17 D. ⌒ — 2 C., axis 25, the corneal astigmatism is 2.50 D., according to the rule, with the axis at 115°. A glass does not improve the vision. The optic disc is a narrow oval with its axis at 115°, grayish-red in color and skirted by an

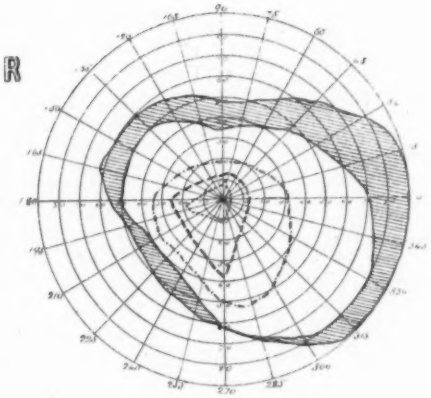

FIG. 5.

Diagram of the field of vision of Case 3, Group 2. Contraction of form and color fields; half defect in the very contracted green field; reversal of the red and blue lines.

atrophic area upon the nasal side twice as wide as the disc. In the left eye the disc is somewhat gray in its deeper layers, but after the correction of a low-grade hypermetropic astigmatism, the vision is normal.

The field of vision upon the right side (Fig. 5) shows contraction for form and colors and again a species of half defect in the very contracted green field, the larger portion of the field being in this instance upon the nasal side. The red and blue lines are reversed.

GROUP III. CASES WITH OR WITHOUT CONCENTRIC CONTRACTION OF THE COLOR AND FORM FIELDS, BUT ASSOCIATED WITH (a) DIMINISHED CENTRAL COLOR PERCEPTION EITHER AT THE POINT OF FIXATION AND SURROUNDING IT, OR BETWEEN IT AND THE BLIND SPOT, OR (b) WITH SCOTOMA, CHIEFLY FOR COLORS.

Case I. M. G., a lad 18 years of age, has no history of squint and no strabismus is now demonstrable. Since childhood he has been amblyopic in the right eye. Vision equals quantitative light perception. The optic disc is a vertical oval with slightly edematous surface. The veins are full and tortuous; macula + 4 D., vertical vessels 7 D. With the ophthalmometer the corneal astig-

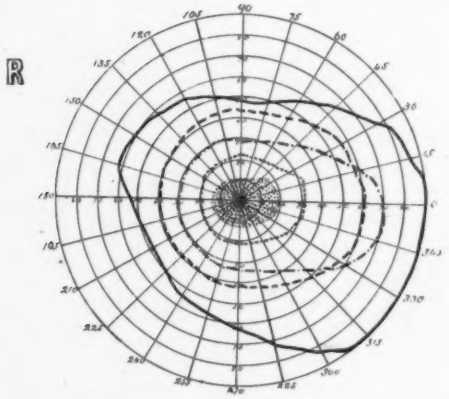

FIG. 6.

Diagram of the field of vision of Case 1, Group 3. Practically no contraction, except in the green field, but a central area of loss of perception of colors with small test objects and weak illumination, indicated by the dotted portion.

matism is 3 diopters according to the rule, with the axis vertical. Correction does not improve the vision. The left eye is normal; refraction low H.

The form field is normal, the red field practically normal; there is slight contraction of the blue field, decided restriction of the green field, and an oval area 35° in its long and 20° in its short diameter, within which there is distinct loss of the perception of form and colors with small test objects and weak illumination, although with large tests object colors are still imperfectly recognized. (Fig 6)

Case II. W. S., a man 27 years of age, has had convergent strabismus of the right eye, with amblyopia, since childhood. The refraction of the right eye is + 2 D. ⌒ + .50 D. C., axis 105, corneal astigmatism being 1 diopter, according to the rule with its axis at 105. The macula is slightly granular, the disc of fairly good color, but there is marked broadening of the scleral ring. All vessels, but especially the veins, are tortuous. The left eye, slightly hypermetropic, is normal.

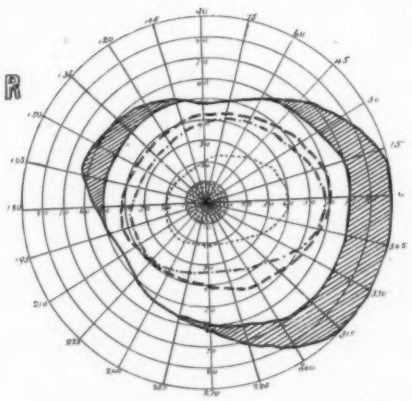

FIG. 7.

Diagram of the field of vision of Case 2, Group 3. Area of diminished color perception similar to that seen in Fig. 6; slight contraction of the form field.

There is slight contraction of the form field; the red field is not far from normal; the blue and green fields are slightly contracted, and there is a circular area surrounding the fixing point for about 10° in which there is marked diminution in the color sense, all colors appearing pale or paler than normal, although with large objects they are still recognized in their true characters. The point at which the color pales is sharply marked from that at which it is perceived in its natural intensity. (Fig. 7.)

Case III. John L., 49 years of age, gives a history of strabismus in childhood, but there is none now demonstrable, nor is there any deviation of the left eye, which has always been amblyopic.

The disc is a vertical oval, the nasal margins are blurred, there is grayness in the deeper layers and marked tortuosity of the veins. The actual refraction is + 5 D. ⌒ + 2 D. C., axis 120, the corneal

astigmatism being 2.50 diopters, according to the rule, with the axis at 120; V. $= \frac{6}{45}$. The refraction of the right eye is H., V. $=$ normal.

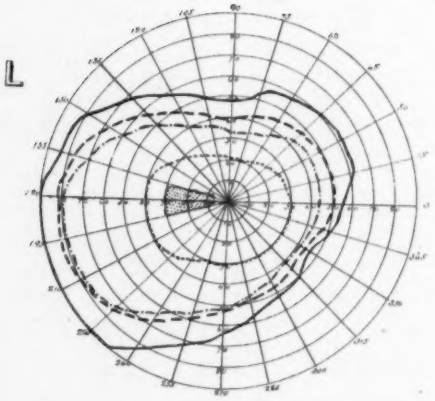

FIG. 8.

Diagram of the field of vision of Case 3, Group 3. Slight contraction of the green field and wedge-shaped scotoma for small colored objects observed under weak illumination, indicated by the dotted portion of the diagram.

The field of vision of the defective eye (Fig. 8) is normal for form and for red and blue. There is slight contraction of the green field, and between 5° and 30° to the temporal side there is a wedge-shaped scotoma for small colored objects observed under weak illumination, although the colors are still recognized as such when the test object is large and the illumination bright.

Case IV. Margaret S., 50 years of age, has had from childhood a "slight cast in the right eye," which was attributed to fright. There is moderate convergence of O. D., vision equals counting fingers at 2 feet; the disc is a vertical oval, gray, the scleral ring broadened all around and the veins full. The refraction is + 3 D. ⊂ + 3 D. C., axis 100. This glass does not improve vision. The left eye presents no important abnormality; refraction a slight myopic astigmatism.

There is marked irregular contraction of the form field of the right eye and a large scotoma for colors passing 20° to the nasal

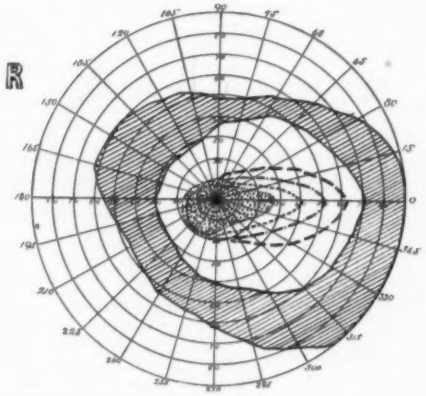

FIG. 9.

Diagram of the field of vision of Case 4, Group 3. Marked contraction of form and color fields and large scotoma for colors indicated by the dotted area in the center of the diagram. There is a species of hemiachromatopsia, colors being chiefly perceived upon the temporal side.

side and nearly 30° to the temporal, 20° below and 10° above. The contraction of the color field is peculiar, blue and green being seen only on the temporal side—again a species of hemiachromatopsia—while red is likewise seen on the temporal side, lost, as are all colors in the scotomatous area, and faintly reappears on the nasal margin of the acquired blind spot. When the patient was last seen there was no change in the vision. (Fig. 9.)

It will be noted:

1. That the cases with normal visual fields and good color perception seem capable of acquiring increased visual acuity, as, for example, Case 1 of the first group.

2. That in the cases with marked abnormalities in the visual fields, particularly in the form of areas of diminished color perception, or color scotomas, there are visible changes in the discs, although in no instance was such an appearance evident as may be seen in the nerve head in toxic amblyopia. Visual acuity in these cases did not improve.

3. That in some cases, for example, Case 1 of Group 3, the abnormality of the visual field is analogous to that seen in hysteria, neurasthenia and allied conditions usually associated with retinal tire.

With regard to the vision of these amblyopic eyes we know:

(a.) The vision of a squinting eye may greatly improve, or the amblyopia entirely disappear, as, for example, in W. B. Johnson's[3] capital case, when for any reason the non-squinting eye becomes suddenly blind, or is removed. The following case bearing upon this point is interesting:

C. S., a man 30 years of age, has had moderate convergent strabismus of O. S. since early childhood, and has never seen well with this eye. About the 1st of November, 1894, the retina of the right, or seeing eye, became detached, and when he presented himself for treatment, December 12. 1894, V. of O. D. equaled counting fingers in the lower field; V. of O. S. $= \frac{6}{15}$, barely, and spells No. 4 at 18 *cm.* with difficulty.

The usual medicinal treatment of retinal detachment—rest in the recumbent posture, pressure bandage and pilocarpin diaphoresis—was pursued, with the result of obtaining in three weeks partial reattachment of the displaced retina, and V. $= \frac{6}{12}$. Vision of the left eye remained, as before, $\frac{6}{15}$. A relapse then occurred, vision of O. D. sinking to about one-third of normal, uncertainly appreciated. The patient declining operative interference, the refractive error of the left eye ($+ 0.75$ S. $\bigcirc + 0.75$ C., axis 30°) was carefully neutralized, and the patient urged to use this eye to the exclusion of the other. In two weeks the vision rose from $\frac{6}{15}$ to $\frac{6}{9}$, and several letters on the $\frac{6}{7.5}$ line. The color field in this eye was normal in all respects.

(b.) The vision of the squinting eye may be improved by exercise,[4] although this is an uncommon record, perhaps, as Randall suggests, "because of the youth of many of the

[3] *Trans. Amer. Ophth. Soc.*, Vol. VI., Part 3, p. 551.

[4] Consult discussion on W. B. Johnson's paper, *loc., cit.*; cases by Risley and Holt, with reference to Javal's case.

patients and the difficulty of applying the tests of vision in such cases," but often, no doubt, because the process is tedious and the results uncertain and discouraging. As every practical ophthalmologist knows, in many cases of anisometropia the visual acuity of the eye with the greater refractive error may be markedly sharpened by exercising its neglected functions, although it is doubtful, as Gould[5] points out, whether this line of practice has received the attention it deserves. While the amblyopia of anisometropia, unassociated with squint, is not strictly germane to the present topic, it at least has some bearing upon the value of exercise and practice with the deficient organ.

If it is true, and it appears to be from such a case as Johnson's, that amblyopia may be due to suppression of the visual image, and furthermore true that such cases rapidly regain vision if required, by reason of the removal or blindness of the fellow eye, to assume the responsibilities of the visual act, is it not worth while to carefully investigate, as Dr. Noyes urges, the field, color field and color sense of these "neglected eyes" and to endeavor to obtain data which might lead to more certain prognostications as to the result of exercising the visual functions than we now possess, at least than the records seem to show that we possess?[6]

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[5] *The Medical News*, December 31, 1892.

[6] I am indebted to my former assistant, Dr. William Bruner, for aid in preparing the diagrams of the visual fields of the cases described in this paper.

SOME FINDINGS CONCERNING SO-CALLED MUSCLE-IMBALANCE AND ITS TREATMENT.

By George M. Gould, A. M., M. D.,
of Philadelphia.

SINCE my suggestion of a method of treating exophoria by prism-gymnastics I have received so many letters concerning some detail or other that I have concluded that a few thoughts derived from added experience in the study of innervational incoordination would perhaps be welcomed by many.

As to the methods of estimating these innervational abnormalisms unless a definite plan of proceeding be adopted confusion inevitably arises. I first of all estimate the static position of rest of the visual axes at 20 feet, and with the Maddox colored rod. This gives the exophoria, esophoria, hyperphoria, or orthophoria, accurately enough for all practical purposes. I do not test the conditions at near range, as I believe all such tests are both comparatively unnecessary and comparatively fallacious.

Next in order is the estimation of the dynamic functions, and the first is the power of abduction, which must be made before that of adduction. For this the improved "double battery of prisms," is a serviceable and sufficient instrument. There is no difficulty about diagnosticating the power of abduction, as it is always definite, sharply limited, and quite uniformly constant. With an abduction of over 8° or 9° one may look for trouble; and the higher it runs the more will be the trouble.

But in the diagnosis of primary adduction many difficulties arise, and different physicians will vary widely in their estimate at the same time and upon the same patient, dependent upon their method of making the test, or upon what is called adduction. Using the "double battery of prisms" I pass slowly from lower to higher prisms, step by step, until one eye slips outward or until diplopia is noted. I find it is best to check the patient's answers by observation of his eyes, as sometimes he will not notice what should be diplopia, but the slipping aside of one eye, or the vibration of the corneas from side to side, tells the observer at once when adduction has been renounced. I then slowly return from the higher powers to the lower until binocular singleness of vision is again reached. One or two sweeps up and down are usually necessary to determine this initial or primary adduction power. It seems to me that it is not properly the highest point to which adduction may be slowly carried from zero upward, nor is it the lowest point at which the eyes slip back into the habitual groove of binocular unity in passing from diplopia toward zero. There is an unnamed neutral zone or boundary line of indefinite and fluctuating limits between the two points. The two points have also not obtained a naming. It is with some trembling that I venture to suggest three coinages. It is premised that the tests are made with prisms, bases out, of equal strengths before each eye, and that the passage from zero to high degrees, or *vice versa*, is by slowly-proceeding, and successive gradations. Proceeding thus from zero upwards:

1. The Upper Diplopsic Point corresponds to that degree of combined prism strengths at which definite diplopia appears.

Proceeding downward from this point again towards zero:

2. The Lower Diplopsic Point corresponds to that degree of combined prism strengths at which binocular singleness of vision (which might be called *binocular monopsia*) is again resumed.

3. The Diplopsic Zone is the space between the upper diplopsic point and the lower diplopsic point.

This nomenclature, you will notice, does not accept the custom-sanctioned method of naming many of our visual

abnormalities *'opias* instead of *'opsias*, derived as the words are from the Greek ὄψις, or ὤψ. It seems outlandish to construct our words, *amblyopia*, *diplopia*, *cyclopia*, *myopia*, etc., as they are written. *Hemianopsia*, *megalopsia*, *micropsia*, etc., are words correctly formed, and it seems to me that we should write *amblyopsia*, *diplopsia*, *cyclopsia*, *monopsia*, because we take an unwarrantable liberty when we "traduce" the Greek letter ψ into our single letter *p*. Our medical myopsic minters should logically have made us say, *eclampia*, *polydipia*, *dropical*, and *droppy*, instead of *eclampsia*, *polydipsia*, *dropsical* and *dropsy*. Perhaps the authors of our 'opia barbarisms thought, if they thought at all, that medical English could hardly be made more contemptible, and if we agree with them we may go on in the old ruts with our 'opeless, myopic, amblyopic 'opias.

I have been in the habit of estimating primary adduction as the middle point of the diplopsic zone, but as this or any estimate of adduction is indefinite and is always varying, it would be more accurate to establish and record the three facts, the upper diplopsic point, and the lower diplopsic point, which con-titute the limits of the diplopsic zone. I believe that the establishment of these factors of ocular motility have not only the interest of theoretic accuracy, but that they have clinical bearings and therapeutic significance.

There yet remains one element of importance in the diagnosis of the dynamic conditions of the eye. This is what I have named convergence-stimulus adduction, a factor the knowledge of which I have found of great practical utility, especially in prognosis. By convergence-stimulus adduction, I mean the limit of 20-foot adductive power gained within a few minutes by successively and rapidly-increased handicapping of the adduction power by prisms, bases out, and in conjunction with intense fixation of the patient's gaze upon an object repeatedly carried from the near point to the distant point.

In young persons with subnormal adduction but with good nervous or mental power, and even in older people under the same conditions, the convergence-stimulus adduction is usually from twice to three times that of simple or primary adduc-

tion. In the neurasthenic and in the aged, convergence-stimulus adduction is much less. The point of practical utility consists in the fact that the extent of the diplopsic zone, or the difference between primary adduction and convergence-stimulus adduction quite accurately forecasts the reaction power of the patient, and thus establishes the probable length of treatment and the general prognosis. The greater the difference the better the prognosis.

The diagnosis of hyperphoria is simple and subject to few doubts. I would only suggest one important addition: The Maddox rod for this purpose should be long and set as closely to the cornea as possible, and then it should be made out clearly whether or not there is any variation in the amount of hyperphoria when the patient's head is raised and lowered as far as possible, while the gaze, of course, is fixed upon the light 20 feet away on a level with the eyes. If there is a variation its amount should be noted. It has significance.

I have found that a small jet of gas-light coming horizontally toward the patient, with a large background of black velvet, makes a brilliant and helpful object-light for all dynamic tests.

Permit me here to interject a word as to a peculiar finding I have stumbled upon in the practical carrying out of prism-gymnastics after the manner I have indicated. It has considerable suggestiveness for the oculist, but it may also be of more or less interest to the general neurologist. It is this: In gazing at the object moved from the near point to the distant point, the patient is able to preserve monopsia and sustain a prism-handicap of a considerably higher degree, if, while sitting still, the object is carried away by another. Diplopia will supervene more quickly during the recession by the mere fact of the patient assuming the standing position, and if the patient attempt walking backward from the unmoved object the innervation previously capable is then no longer obtainable. Sometimes, even, the patient cannot bring about monopsia with a handicap previously borne at 20 feet while sitting, but in standing and walking toward the object it is impossible, although at the closest range and with the most powerful effort. This fact, therefore, is

the exact reverse of that known as the Reinforcement of Reflexes. In extreme ocular convergence-stimulus adduction synchronous innervation of the muscles of the body lessens and destroys the highest ocular adduction power instead of increasing it. I have even noticed that the jar of a definite step will destroy the delicate equilibrium, when it was held by a soft and sliding step backward.

In order, therefore, to gain a complete expression of the dynamic functions of the eyes we must obtain knowledge of seven factors:

1. The lateral "Phoria,"—*i. e.*, the lateral orthophoria, the esophoria, or the exophoria.
2. The vertical "Phoria,"—*i. e.*, the vertical orthophoria, the hypophoria, or the hyperphoria.
3. Abduction.
4. The Upper Diplopsic Point. } The Diplopsic Zone.
5. The Lower Diplopsic Point. }
6. Primary (or simple) Adduction.
7. Secondary (or convergence-stimulus) Adduction.

With a statement of these seven elements, together, of course, with the diagnosis of the refractive error, one who has never seen the patient may gain a pretty accurate knowledge of the prognosis and therapeutics of a given case of functional ocular disease.

The question arises: What is Orthophoria? The answer of the distinguished and honored originator of the word is well known. But we must be on our guard against the unconscious admission that orthophoria is normal, healthy, or the best sort of "phoria." My experience is that a person with orthophoria, *i. e.*, one whose visual axes in a condition of relaxation and rest meet upon an object 20 feet away is usually in an unphysiologic working condition if he has much near-range work to do. Orthophoria is generally a poor sort of 'phoria; it is functional disease; it is in a sense not orthophoria. Our urban slaves of civilization, the severe ocular laborers, need that excess of adduction power that we call esophoria, or its equivalent, to insure the best physiologic function. Esophoria of from 2° to 5° is thus, again in a sense, orthophoria.

But in the light of my experience the terms orthophoria, esophoria, and exophoria, are often inexact and misleading,

always unsatisfying, and, standing alone, they signify little or nothing. Although compelled to use them they are of no use to me except as coupled with the other six factors I have enumerated. One patient with "orthophoria" may need no treatment, whilst another with the same condition may need long treatment. It all depends upon the other factors, and especially upon the relation between abduction and the two adductions, primary and secondary.

In the first place treatment of muscular anomalies should depend upon two other things:

1. It should usually depend upon the existence of symptoms ascribable to the dynamic strain. Nature has so many subtle ways of compensation and of self-cure, that we are grossly impertinent if without symptoms we rush in with our blundering devices and our asinarian therapeutics. The antics of some drug-maniacs and operation-madmen seem to rest upon the assumption that God has but little wit, and that He needs much instruction in physiologic matters. Let us at least wait for some sign, such as pain, asthenopia, etc., that He has failed.

2. Treatment should also depend upon the thorough trial of correction of the refraction error. No living man with a vestige of native modesty is at all sure that he knows much about the precise etiologic relations between errors of refraction and errors of motor-coordination, but every observant and intelligent student is thoroughly convinced that, in a general way, refraction-errors are the vastly preponderant source of eye-strain. It, therefore, behooves us to first see if accurate correction of the ametropia will not bring relief, and perhaps superinduce comfortable muscular function. If it does neither then may we proceed with our endeavors to learn other causes of discomfort, and to institute the means of relief.

As to the treatment of esophoria, the hope has proved fallacious that I at one time entertained that a reversal of the method that had proved so successful in exophoria would be of similar service in esophoria. But in learning this lesson I have learned another, and that is that usually with proper correction of the ametropia, esophoria as a rule ceases to become trouble-

some, or is gradually cured by the correction of the refraction-error. This especial lesson, however, so far as concerns esophoria, it must be confessed, is based upon a very limited experience. While I have had in the last years perhaps several hundred cases of exophoria, I have not had, I guess, over a dozen cases of esophoria. One, though not my own, yet personally known to me, was a dismal failure. The internal recti tendons (not by myself) were both cut clean off on two separate occasions, with a prompt return in a few weeks of the total (16° to 20°) esophoric imbalance, the sleepiness and the asthenopia. Another patient, when last seen, had about 18° of esophoria without the slightest symptom of inconvenience. My other cases, as I have said, either find no inconvenience from moderate degrees of esophoria, or the normal balance of the muscles under proper glasses is being certainly and progressively established.

As to hyperphoria, a similar result of the use of correct spectacles is usually seen, and this result is hastened by prism-gymnastics, handicapping the tendency to binocular vision and arousing an increase of effort by the will, and by extreme motility of the eyes upward and downward, the head being kept rigid.

When the hyperphoria is high, I am accustomed to correct only a part of it in the spectacles by prisms, leaving an unreached ideal for the nervous mechanism to strive toward. In one case, the highest degree of non-traumatic hyperphoria I have ever seen, there was a defect of 15°. The man had visited several oculists, and had about made up his unwilling mind to submit to tenotomization. The diplopia, headache, etc., were intolerable. I gave him a proper correction of his ametropia, which he had never had, with a correction of two-thirds of his hyperphoria, which produced "monopsia" at once, and a most exuberant satisfaction and gratitude; I also instructed him in simple methods of ocular gymnastics. In a short time the 5° of uncorrected hyperphoria had about disappeared, and I expect at his next visit to halve the hyperphoric prismatic correction.

As to the treatment of exophoria, or as I prefer to call it, of subnormal adduction, I have nothing to recant and little to modify. The plan previously suggested has proved entirely

satisfactory. And this is all the more satisfying because the vast majority of all so-called muscle-troubles are of this class. I have not had time to gather accurate satistics, but I would judge that fully 95% of all my cases of "phoria" requiring treatment have been cases of so-called exophoria. I consider the method applicable whenever—even in cases of divergent strabismus—there is any tendency, or power whatever, toward binocular fusion. Whenever there is (convergent) life there is (binocular) hope. It is wonderful what marvels of therapeutics nature will bring about if we give her only a wee bit of help here, remove a little obstacle there, and if we arouse effort and instil persistent patience everywhere.

The essence of the matter in all these cases of so-called exophoria is to increase the adduction-power. A fig for this fetich of exophoria! Bring the habitual power of adduction sufficiently high and the "exophoria" may be left to care for itself; both it and the symptoms will soon disappear. Here, in fact, lies one of the troublesome things about the method. When the symptoms disappear the patients cease coming, thinking they are "cured"—when they are only temporarily relieved. Quite a number of patients, certainly not at my suggestion, and against my protest, keep their prisms for "emergencies," and whenever, from overuse, ill-health, or from any cause whatsoever, headache or other symptoms recur they use them for awhile with speedy relief.

The proper way to do is to carry the adduction-power so high, and fix it there by continued gymnastics so permanently, that the treatment may once for all be discontinued. The adduction-power has sometimes to be carried very high indeed before this safe point is reached. I have had a number of patients in whom it was brought to 80°, in several to 90°, and in two to 100° or 110°.

In no other class of cases is it so impossible to lay down rules as in these so-called, wrongly-called, muscle-cases. Every case becomes a study in which all treatment must be guided by careful and prudent intelligence, applied to the purely individual and ever-varying condition.

The fundamental principle underlying this non-operative gymnastic therapeutics of muscular imbalances is the conception of the eye as a living, self-healing, marvelously delicate,

dynamic mechanism. Our aim should be to remove obstacles and provide helps for it in its struggle to do the awful work thrust suddenly upon it, of civilization, with an instrument not prepared, habited, or fitted for that work. Our single aim is to provide helps so that it may do its labor physiologically. We must utterly root out of our minds the odiously wooden-headed assumption that the eye is a dead, rigid, and mechanic system, and that its muscles and their tendons are like taut wires moved by a sort of stupid clockwork machinery from behind and needing constant tinkering to make them a little shorter or longer. The ocular dynamic apparatus is a living organ, changeable, keenly alert and marvelously reactive to stimulus and to intelligent help. I can imagine nothing more "*bête*," more unsurgical or more unmedical, than cutting the tendon of an overstrong muscle of the eye, when the weak antagonist, by physiologic methods, may in a week, a fortnight, or a month, have its strength increased five or six fold.

CHRONIC INTERSTITIAL OPHTHALMITIS.
(CHRONIC SIMPLE GLAUCOMA.)

By S. O. Richey, M. D.,
OF WASHINGTON, D. C.

THE reader of the literature of glaucoma must be impressed with the regional consideration given to the subject by all writers, since the announcement of Von Graefe's high-tension theory. The whole process, and its explanation, have been confined to the ocular structures alone. In 1892,[1] the writer attempted to show *why* glaucoma should be regarded as a local expression of a diathesis. One of the ablest advocates of the retention theory (Knies), of increased intra-ocular tension, Priestley Smith, thinks "*that the high tension depends more upon an excess of blood in the eye than upon an excess of intra-ocular fluid.*" In favor of this is the *rapid* advancement of the lens and iris in acute glaucoma; the engorgement of the venal system; and the pulsation of the retinal arteries, intensified by slight pressure upon the globe; thus showing increased general arterial tension, accentuated by the added resistence to the entrance of blood into the eye.

High tension of the bulb is not glaucoma, but with the other symptoms of glaucoma, is a localized expression of a general affection, which is aggravated by violent emotion, by shock, by excess of food and indigestion, by hunger, by loss of sleep, by the climacteric, by constipation, and by palpitation of the heart; just such disturbances as aggravate this disease

[1] *Trans. American Ophthal. Society*, 1892.

expressed elsewhere in the system. Such passing conditions do not *cause* glaucoma, but they precipitate the attacks when a predisposition already exists, and the *modifications* of tension can be more readily understood, attributed to this factor, than when referred to either of the most prominent theories of high tension, that of hypersecretion, and that of retention, of the intra-ocular fluids, to each of which fatal objections exist. For, the mechanical resistance of the eyeball excludes the possibility of hypersecretion beyond the point at which the two would balance each other; and this objection stands equally against the theory of retention, as the secretion of fluids can not be continued beyond a certain fullness of the globe, which is reached more quickly if the secretions are retained. Also, the channels of excretion are often found open after the highest degree of tension, which takes place in the acute form of glaucoma, when the heart's action is usually excitedly strong, in its effort to overcome spasmodic contraction of the remote arterioles; a condition shown by the pallor of the face, followed by dilatation of the vessels and congestion of the face and head, with slight protrusion of the eyeball, and some edema of the lids, the condition in which we usually first see the cases.

The bilateral character of glaucoma must be remembered, for in nearly all cases, sooner or later, both eyes are affected in this way. Attention has already been called to its close resemblance to another local expression of the same constitutional malady in another extremity of the body, of which high tension is as constant a peculiarity, and which is accepted as due to turgescence of the blood vessels. Von Graefe believed all forms of glaucoma to have one cause, which he judged to be local, increased intra-ocular tension; by which the attempt has been made to account for all the other phases of the disease, local and general, symptomatic and anatomic; hence, much of the confusion.

Corneal opacity, which has by no means been ignored, as the reports of the vast majority of cases mention it, has not had due prominence, nor the importance attached to it, which its constant presence deserves. It is, perhaps, more constant than increased intra-ocular tension, which it often precedes;

and, in such instances can no more be due to increased tension than can disc excavation in a case in which tension has never been above normal.

To take a lesson from the affection, and begin at the beginning, is a course which is simple and logical. Donders indicated the beginning when he declared chronic simple glaucoma to be *the type* of the disease. The other forms of glaucoma have the same origin, the cause acting with a varying degree of force or virulence. Chronic simple glaucoma, the primary and uncomplicated form, is the initial localization of the process, from which arise all other forms and complications. It may be so slight that its known symptoms are not recognizable, for the disease has often developed beyond the first stage, lacking most of the diagnostic symptoms, until an added impulse has declared them; and yet, in nearly every case, there is an antecedent history of headaches, called neuralgic, which seem to start from the occiput, like the headache due to breathing coal gas. To quote Foster:[2]

"The dominating center of the vaso-motor nerves lies in the upper floor of the medulla oblongata. Stimulation of this central area causes contraction of all the arteries and great increase of arterial blood-pressure, with swelling of the veins and heart. Paralysis causes dilatation of the arteries and fall of blood pressure. This center may be excited directly and reflexly. * * * Psychical excitement influences this center, causing constriction of the small arteries. * * * A pale, cold, collapsed side of the face, contraction of the temporal artery like a whip-cord, *dilatation of the pupil*, and secretion of thick saliva, are sure signs of intense stimulation of the cervical sympathetic nerve, which may be brought about by some poisons, and by emotion. Lactic acid (1-10,000 saline solution) passed through the blood vessels of a frog always enlarges their caliber (Gaskell)."

Simple glaucoma has fairly constant features. Acute glaucoma is of many varieties, whose features in common are increased intra-ocular tension, advancement of the lens and iris, dilatation of the pupil, and opacity of the cornea; the variation depending upon the quantity of impulse, the special tissues involved, or upon the stage which has been reached by the chronic process, on which the acute form is engrafted.

[2] Foster's Text-book on Physiology, second edition, pp. 695-701.

That the acute form is propagated upon a local process already existing,* and more or less entangling the tissues of the whole organ, would seem to be supported by the irregularity and uncertainty of its development and response to measures adopted for its relief. The anatomic and microscopic changes noted, in the different series of glaucomatous eyes examined, may disagree; at times they directly contradict each other. This is not due to incapable or imperfect observation, though it causes great confusion in the mind of the student; but to the fact that the underlying process has attacked a different set of tissues, or the same tissues in varying order; has not pursued an identical course in its encroachments, or has done so with diversity of power or rapidity. Observations have included all the tissues of the eyeball. In one case, at a given period of development, the disc will be deeply cupped, while in another the excavation may be shallow, if it exists; the anterior chamber may be shallow in one case, and deep in another; tension may be the same in all; or with any, or all, of these changes it may not be increased. The sinus of the anterior chamber may be closed with normal or subnormal tension[3]; in another, aniridia[4], with the filtration angle closed by the *rudimentary* iris, and with increased tension. Any combination of symptoms and conditions existing together point to two processes at work: one, chronic and constantly progressive, the other spending itself in recurring attacks of more or less violence; both due to the same influence, which manifests itself in the two ways, in different degrees, in individual cases. A remote nervous influence manifests itself when mental perturbation excites an exacerbation, or when rest and quiet are followed by an improvement in the physical symptoms.

Whatever other changes are observed, those of the cornea are nearly always present. That glaucoma may begin in the cornea there is hardly room for doubt, for repeated slight injuries to the cornea are frequently followed by acute glaucoma, as is shown by the interesting cases given below in

* May not always be apparent.

[3] Schnabel, *Archiv. Ophthal.*, Vol. VII., pp. 37, 38, 302.

[4] Collins reports several cases, *Ophthal. Review*, Vol. X., p. 101.

abstract; even two of Mr. Collins' three cases of aniridia with glaucoma were marked with initial corneal changes; one with central leucoma, consecutive to a perforating ulcer of the cornea, and a staphyloma in the ciliary region; another, with a corneal cicatrix and a staphyloma; the third had a glaucomatous cataract at 34 years of age.

Von Graefe[5] reports a woman 50 years of age, with an old eczema for many years, with an opaque and swollen spot with a yellow center, in the cornea opposite the pupil. Tension, which was not tested at the time of his first interview, was normal in the fourth and sixth week. While the corneal disease remained stationary, a subacute glaucoma developed. Iridectomy relieved the glaucoma and cured the corneal affection.

Saemisch[6] relates a case, in which a *striped* opacity of the cornea existed previous to the development of a vesicle. When the corneal trouble was at its worst, acute glaucoma supervened. Iridectomy; cure.

Pooley[7] observed a case of keratitis vesiculosa, ending in absolute glaucoma. A Jewess, 40 years of age, left eye. The anterior chamber, fundus and field normal, V. = $\frac{20}{50}$, in August. In October, pupil wide and immovable, anterior chamber shallow, T (r) +, no perception of light, great pain. Iridectomy reduced tension and relieved the pain, but did not restore vision.

Saemisch[8] gives another case, blind from glaucoma, in which corneal vesicles developed. The eye had to be enucleated.

Bowman reports a case of keratitis bullosa in an ill-fed woman; the eye was destroyed by glaucoma.

Landesberg[9] details seven cases of keratitis bullosa, illustrative in this connection:

Case I. A child 8 years of age, in whom severe ciliary neuralgia, and increased intra-ocular tension, accompanied the development of each vesicle, which occurred at intervals of from eight to fifteen days. Reparation began in the deeper layers of the cornea.

Case II. A man 38 years of age; the corneal affection pursued the same course for four months, ending in acute glaucoma, cured

[5] *Archiv. f. Ophth.*, Vol. XV., p. 108.
[6] *Berliner Klinische Wochenschrift*, No. 37, p. 449.
[7] *Archiv. Ophthal.*, Vol. IV., p. 46.
[8] *Handbuch der gesammten Augenheilk.*
[9] *Archiv. Ophthal.*, Vol. VI. p. 135.

by iridectomy. His family physician judged this man's trouble to be of *rheumatic** origin.

Landesberg thinks there is such an interdependence between keratitis bullosa and glaucoma that every eye suffering from this form of corneal trouble is liable to be seized with glaucoma.

Case III. In a man 22 years of age, ulceration of the cornea, consecutive to a foreign body (bit of metal) in the cornea, in October. Vesicles formed six times before the end of December, always announced by the presence of several small ulcerations in the deeper layers of the cornea, from which vertical parallel *stripes* proceeded. In four of the six attacks increase of tension was evident.

Case IV. A child, four years of age, with *eczema* of the head, nose and face, had frequent attacks of keratitis bullosa; irritation, pinhead infiltrations in the deeper layers of the cornea, from which proceeded vertical *stripes;* ciliary neuralgia and increased intra-ocular tension. The eye recovered with a circumscribed leucoma.

Case V. A man, 36 years of age, had frequent attacks of keratitis bullosa (seven to fifteen days interval), with the already described appearance of the cornea, and increased intra-ocular tension. He recovered with a firm cicatrix at the site of the affection.

Case VI. A man, 71 years of age, with a like appearance and condition of the cornea, anterior chamber shallow, pupil moderately dilated, increased (?) tension. Nine days later, a vesicle; acute glaucoma; iridectomy. Two weeks later, another vesicle, tension increased. Cured, with a circumscribed central leucoma.

Case VII. A girl, 20 years of age, without previous trouble. The appearance of the cornea as in the cases described; tension normal. Three weeks later, vesicles formed with increased tension. A number of attacks followed in the next four months, when the patient discontinued her visits. At the last visit, the lower part of the cornea was dim, and tension was normal.

Atropin was used in all these cases, but atropin will not *cause* increase of tension in a normal eye. A disposition to high tension must pre-exist, and its occurrence is only precipitated by the mydriatic; possibly by the retraction of the iris,† and the consequent diminished resistance to the influx

* The identity of the cause of gout and rheumatism can not be discussed here.

† Eserin acts by resisting this influx; when the *vis a tergo* is too great or too persistent, it fails.

of blood into the eye in the presence of general high arterial pressure, as shown by the tendency to intra-ocular hemorrhage if too rapid escape of aqueous is permitted during glaucoma operations. Landesberg found the best treatment of these cases to be by *scarification* and *compression*. He refers to the very rapid onset and course of the corneal phenomena, save the opacity; to the increased intra-ocular tension, which is constantly present and in proportion to the corneal irritation; to the immunity of the iris and choroid, and to the secondary (?) glaucoma.

Bullous keratitis, according to Graefe, *is one of the symptoms of a deep morbid process.* In its nature it is a herpes, and not of true inflammatory character; it is an expression of irritation at the origin, or in the course, of a nerve, an evidence of which we have in the neuralgic pain inseparable from the development of the vesicles. The periodicity of its recurrence (seven to fifteen days) suggests an irritation of chronic character, and in this is supported by its apparent kinship to herpes preputialis, which has the same history, expresses itself in the same manner, runs the same course, and of whose origin there is no doubt; lithemia. The vesicles may precede the development of glaucoma, and be of some prognostic import; they may develop only in the stage of glaucoma degeneration. Appearing thus at widely different stages of this disease, the two must be due to the same cause, and form part of the same process.

Landesberg[10] further relates two cases of *ribbon-shaped* keratitis (?) followed by glaucoma, which are not without interest.

Case I. "A man, 60 years of age. When first seen, the right cornea was the site of a wide ribbon-shaped and continuous opacity, corresponding to the palpebral fissure, and extending transversely over the whole cornea. The opacity was a *reddish-brown*, and had an equal degree of color intensity from the margins to the center of the cornea. The center itself appeared whitish-gray, as if a metallic salt had been precipitated there. The ribbon-shaped opacity had a sharply defined boundary above and below." Photophobia, tension normal, pupil reacted sluggishly to atropia; ophthalmoscopic exam-

[10] *Archiv. Ophthal.*, Vol. III., p. 65.

ination impossible. The left eye showed the first stage of the affection; near the inner and outer margins of the cornea, a narrow opaque stripe of slightly brown shade. The opacity had the appearance of a thin stripe of brown color, laid quite superficially on either side of the cornea with the most delicate touch of a brush; the center of the cornea clear. He rejected an iridectomy on the right. Three months later he returned. The right eye was *hard*, with a shallow anterior chamber, posterior synechia, perception of light, subconjunctival injection; corneal opacity unchanged. Iridectomy with good result. Three years later, there was no irritability, tension normal, and the corneal opacity had a tendinous appearance, having gained so much as to leave a very narrow rim of clear cornea.

Case II. A man, 55 years of age. When he presented himself, the right eye was the seat of subacute glaucoma. All the phenomena of glaucoma were present, with numerous hemorrhagic spots in the retina. The cornea was affected with ribbon-shaped opacities, brown in color; the corneal epithelium was smooth and shining. Iridectomy reduced the tension and cleared up the hemorrhagic spots, and the eye remained in a satisfactory condition, with no gain in the ribbon-shaped opacity, a year later.

Hirschberg[11] records a case of acute glaucoma, following the use of atropin, in "an apparently healthy lady," 64 years of age. He found "fine maculæ cornearum centrales *of old date* and punctate opacities of the front surface of the cornea." Atropin was used after iridectomy without mischief, and H. raises the question if glaucoma malignum *belongs to the eye or the individual.*

In but few of the cases cited above was the cause suggested for the corneal changes observed. A notable exception was that in which a bit of metal excited the changes which followed; and yet a constitutional predisposition existed, or the formation of vesicles would occur more frequently from foreign bodies in the cornea. This predisposition was probably "rheumatic," as suggested by the physician in another of the cases.

The cornea may be measurably tolerant of injury, but just what persistent mechanical irritation will accomplish (if that alone did it) is shown by the cases reported by Dr. Hock,[12] of Vienna, almost in the nature of an experiment.

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[11] *Archiv. Ophthal.*, Vol. IV., p. 203.

[12] *Archiv. Ophthal.*, Vol. V., p. 382.

"A right eye had been lost six months previously from Egyptian ophthalmia, leaving a dense cicatrix in place of the cornea. Fingers could be counted at 2 feet, tension normal. The cornea was tatooed, and during the evening there was severe pain on the right side of the head, extending from the eye. Next day the bulb was of stony hardness. A former corneal fistula was reopened, and the globe immediately became quite soft. Tension was increased four times during the treatment; each time the lens was advanced, with bulging of the middle part of the scars.

"The curative effect of artificial interference after each relapse (opening the fistula), and especially the recovery and persistence of normal tension after the last sitting (when the fistula was again reopened) proves to me that in this eye there were none of the conditions (!) predisposing to glaucoma, but that the effect was due entirely to the multitude of little wounds."

"Irritation of the cornea is sufficient, without mediation, to give rise to glaucomatous increase of tension" (Von Gräefe). Hock adds the case of a stout woman, 54 years of age, with palpitation of the heart, whose right eye had been lost a year before by glaucoma. "Just below the middle of the cornea was found a circular opacity as large as a hemp seed. Evidently there had been circumscribed corneal infiltration preceeding the glaucoma which had now passed by. Considering all the circumstances developed by the history of the right eye, we can hardly avoid the conclusion that here was a succession of corneal infiltrations, with consequent glaucoma."

Mauthner[13]: A man, 71 years of age, injured by powder explosion a long time previously. In youth, he had some inflammation, which left several facets as large as a pin's head, stretching across the area of the pupil. Acute glaucoma of the right eye, with remarkable *striped* haziness of the corneal parenchyma; left eye hazy from an *old* parenchymatous grayish-white and striped cloudiness. Sclerotomy, and the stripes became squares, so that forty-eight hours later the cornea looked like a chess board, the stripes giving way to squares, which disappeared in a week. Fourteen days after operation the cornea was perfectly transparent except for the old central facets; cured.

Mauthner (*Loc.cit.*): A man, 64 years of age. Glaucoma simplex. Both eyes had been struck by the bowstring of a mouse trap a year before. Both corneæ faceted; pterygium covers the pupil of the right eye; circular abrasion of the left cornea, upward and outward.

[13] *Loc. Cit.*, Vol. VII., p. 224.

Tension moderately increased. Iridectomy of the right eye; sclerotomy of the left. Tension notably lessened.

Mauthner (*Loc. cit.*, p. 235): Man, 74 years of age, with both corneæ cloudy in distinct parenchymatous spots, which look like the remains of previous inflammation. Increased tension. R. V. = ½; L. V. = ⅛; F. F. free. Sclerotomy. Tension normal; spots in the cornea unaltered; vision unchanged.

Idem: Boy, 6 years of age; blow on the head; traumatic cataract of the left eye and glaucoma of the right eye. T. + 3, globe very large. Cornea cloudy in *stripes*, diameter of the cornea = 18 *mm.*, disc deeply cupped; no pain; no perception of light. Sclerotomy. T —; fingers at 10 feet. Left cornea clear, diameter = 15 *mm.* (3 *mm. less* than that of the glaucomatous eye).

Schöler[14] produced glaucoma experimentally by burning the cornea. This was probably due to the irritation of the cornea, and the consequent disturbance of the normal supply of corneal nutritive elements or to reflex nerve influence upon the vaso-motor "dominating center" in the floor of the medulla.

Dr. Elizabeth Sargent[15] made an anatomical dissection of six eyes enucleated for absolute glaucoma. She found *pannus of the cornea in all;* superficial ulceration in one; masses of round cells about the scleral and episcleral vessels, or in the cornea, in all; sinus of the anterior chamber patent in three; closed in two; partly open and partly obstructed in one; iris atrophic, and adherent to the cornea, in all; Descemet's membrane pierced by inflammatory tissue (showing *how* this membrane may not *always* be intact), in one; retina atrophic, and detached, in three; with sclerosis of the arterial walls in three; optic nerve excavated, with connective tissue hypertrophy, or celular infiltration of the nerve, and atrophy of the nerve, in all.

It is with difficulty that an abstract is made of such a detailed examination and reference is made to the paper. The examination was very pains-taking and offers evidence of the constancy with which corneal structure changes, cellular infiltration into the corneo-scleral region, and into the optic nerve and its sheath, and connective tissue hypertrophy, are met. These are the ultimate features of the structure changes,

[14] *Berliner Phys. Gesellschaft*, June 20, 1879.

[15] *Centralb. für Augenheilk.*, December, 1884, p. 353.

for, in these cases, the malady had already run its course. To specify all the changes that had taken place at any particular period of its progress is manifestly impossible, as they can not be just the same in any two cases, and thus give rise to a diversity in the severity of the symptoms, and of the result of a given operation, in different cases. This examination seems to have been made with no purpose to support a preconceived theory, and is reliable.

The examination of seven glaucomatous eyes by Birnbacher and Czermak [16] is of much the same character as the preceding, only it shows how persistently an accepted view possesses the mind. The four, first in order, had chronic inflammatory glaucoma; the next was one of hemorrhagic glaucoma; the last two, of glaucoma degeneration.

Case I. Cornea rough and exfoliated; T. + 2; slight edema of the corneal structure; slight rupture of Bowman s membrane, and two or three-fold layers of spindle cells between it and the pavement cells; spindle-form, and large groups of round cells, with single epitheloid cell forms about the episcleral veins. Cell infiltration into Schlemm's canal, membrane of Descemet, iris, ciliary body, choroid, in the sclerotico-choroid about the papilla; and masses of them rest in the perivascular spaces, and take on the form of young granulation tissue. The choroid is thinned about the papilla. An exuberant growth of endo-epithelium was found everywhere in the intervaginal space.

Case II. Retina and choroid adherent in places. Pavement cells of the cornea destroyed. Bowman's membrane broken through, and epithelium thickened. In the upper layer of the corneal structure, and in Bowman's membrane, was found a thick, fibrous, richly nucleated tissue, having vessels, and penetrating the surface of the membrane, always at least to the extent of the area of the membrane. This tissue was found between Bowman's membrane and the epithelium, and in the epithelium, causing small *elevations* of its surface. Blood vessels extended to the center of the cornea, and along them thick rows of round and spindle cell nuclei. The canal of Schlemm was effaced. Fibrous connective tissue was found on the surface of the iris; the retina, posterior to the ora serrata *was greatly thickened, and changed into an irregular meshwork of course and fine fibers;* the posterior choroid thin and adherent to the

[16] *Graefe's Archives*, Vol. XXXII, 2, pp. 1 to 75.

sclera; nuclei of the optic nerve neuroglia increased in number; about the central vein a mass of granulation cells.

Case III. Relation and position of the cells of the pavement epithelium of the cornea changed. No edema. Between the corneal epithelium and Bowman's membrane was found a tissue of coarse fiber bundles, and numerous long spindle-form nuclei, supplied sparsely with vessels, and extending over the whole cornea. Bowman's membrane was broken through in many places by lines of spindle cells, which sink into the corneal structure. Scleral and epi-scleral vessels extended forward through all the layers. The corneo-scleral sinus was almost obliterated. In the anterior section of the corpus vitrei were numerous wander-cells.

Case IV. A hemp-seed sized sclerectasia; in the corneal epithelium were circumscribed small prominences; between Bowman's membrane and the epithelium the space was filled with a fine mass of *striped* network. Slight edema.

Case V. In the corneal substance, especially near the periphery, and in the deeper layers was found a coarse *network* whose contents were invisible: membrane of Descemet intact.

Case VI. Edema of the cornea, and colored nuclei. Bowman's membrane broken through by spindle-shaped cells.

Case VII. A woman, 75 years of age, with high arterio-sclerosis and irregular heart action; urine normal. Pericorneal injection, keratitis punctata, cornea uneven, anterior chamber abolished. In the cornea fine granulation masses were found in the epithelial spaces. Glaucoma pannus; spindle-celled formation in the corneal substance.

It will be seen that the new-formed connective tissue was found in all these cases and in more than one structure, but especially marked and general in the cornea.

I have herein referred to no cases of my own, for, in the language of Mooren, "I prefer to call attention to the statements of other observers rather than to mention cases of my own practice, in order to leave no room for the insinuation that my observations are biased in favor of a certain pathological view, thus bringing into connection with each other things which may be only the result of accident."

I would say further, that, for obvious reasons, I have avoided reference to the "steaminess" of the cornea, which is always manifest in acute glaucoma, and which may, or may not, be due to edema of the cornea from increased intra-

ocular tension, as claimed by Fuchs[17]; nor to the arcus senilis (the early result of the affection of the corneal nutrient vessels, as the ulceration, or necrosis, of glaucoma degeneration is the late development) so often present in glaucoma; nor to the lenticular changes, though Rheindorf describes glaucoma with *acute* opacity of the lens, the cataract being incipient when the glaucoma developed. I would suggest that a closer relation exists than is realized, between the *cause* of glaucoma and the *cause* of senile cataract.

Angelucci[18] thinks that glaucoma is due to sclerosis of all the membranes of the eye, and especially of the walls of the blood vessels, as he found the arteries sclerosed, their caliber diminished, and the veins swollen and chronically inflamed.

Steamy opacity of the cornea may precede increased intraocular tension, and two weeks may be required for its disappearance after tension is reduced. It is an open question, if fluid from the anterior chamber can penetrate to the corneal lamina while the membrane of Descemet remains uninjured. The cases referred to show true corneal tissue changes, often of some duration when the glaucomatous attack developed: stripes, brown ribbon shapes with smooth and shining corneal epithelium, chess-board squares, vesicles, facets and leucoma; which differ greatly from the possible appearance of an intralaminar edema, with rough and desquamating corneal epithelium. Fuchs and others have seen intralaminar edema, and it, therefore, sometimes occurs, but only when the membrane is deficient at some point, as in Sargent's case, in which Descemet's membrane was perforated by inflammatory tissue. A few cases, however, do not show that the corneal opacity is due to edema from high tension. It *may* happen so, and another form of defficiency in the membrane of Descemet by which edema of the cornea from the interior of the eye may occur is shown by Tartuferi,[19] who, in a microscopic examination of the corneæ of a collection of glaucomatous eyes, found in most of them tissue changes of important character: shortening of the corneal diameter by connective tissue between

[17] *Graefe's Archives*, Vol. XXVII., 3, p. 66.

[18] Trans. Internat. Med. Cong., London, 1883.

[19] *Giorn. di R. Acad. di Med. di Torino*, Nos. 5 and 6, 1882.

the epithelium and the membrane in the periphery of the cornea; the presence of wander-cells which are easily changed into connective tissue; the epithelium often separated from Descemet's membrane by connective tissue, etc.

Here again we find corneal changes which are of the same character as those noted in the other structures of the glaucomatous eye, for by anatomical and microscopical examinations connective tissue has been found in the ciliary nerves[20], muscle,[21] and processes,[22] the cornea,[23] the iris,[24] the choroid[24], the retina,[25] and the optic nerve.[26] Fuchs,[27] himself, from the dissection of the eyes of a woman, dead at 64 years of age, which had been iridectomized for acute glaucoma eight years before, in which the lamina cribrosa was only slightly convex backward, with atrophy of one-fourth of the optic nerve fibers, concludes that simple hyperplasia accounts for the changes in the ciliary body, as they seem to have existed *before* the glaucoma, and to have excited the attack. Connective tissue hypertrophy was most marked in the region of the macula.

Schnabel[28] thinks opacities of the cornea do not depend upon increased tension, and that they are only a symptom; that they are not of inflammatory character in the sense of a keratitis, differing so much in appearance, some being dotted, some ribbon-shaped, and others diffuse, occupying the whole corneal tissue. Yet, some of them have as much of the character of inflammation as the so-called inflammatory glaucoma; we do not see *pus* associated with either. It is true that ulcers and abscesses of the cornea in absolute glaucoma, are sometimes accompanied by hypopion, but only as

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[20] Hocquard, *Archiv. d'Ophth.*, Vol. III., No. 3

[21] Stolting, *Graefe's Archiv. f. Ophth.*, Vol. XXXIV.

[22] Brailey, *Royal Lond. Ophth. Hosp. Reports*, Vol. X., p. 86.

[23] Birnbacher and Czermak, *Graefe's Archiv.*, Vol. XXXII, 2.

[24] Knies, Ueber das Glaucom., *Archiv. f. Ophth.*, Vol. XXII., p. 163.

[25] Schnabel, *Archiv. Ophthal.*, Vol. VII., p. 307.

[26] Alt, Lectures on the Human Eye, p. 156.

[27] *Graefe's Archiv.*, Vol. XXX., p. 123.

[28] *Wien. Med. Presse*, 22-26.

a result of necrosis, for the glaucomatous character of the affection has about ceased. It would, therefore, seem proper to attach to the corneal changes, observed in glaucoma, their true importance as *part of the process* instead of trying to account for them as a result of increased tension, a superficial view which the evidence does not justify. If, however, they *must* be regarded as a symptom, let it be as a symptom manifested in the cornea, exposed to view, of a tissue change progressing elsewhere in the structure of the eye, out of view; in the sclerotic. The epi-scleritis, which often foreshadows a glaucoma, favors this view.

Dr. J. Kostenitch[29] reports the microscopic examination of a case of scleritis:

"A woman, 24 years of age, left eye blind, *larger* than the right and sensitive to pressure; Tn. Entire cornea a white scar. Optic disc excavated; anterior chamber shallow; iris atrophic, and adherent to the cornea at its periphery. Sclera, cornea, conjunctiva, iris, ciliary body and vitreous affected by cellular infiltration, and occupied by numbers of wander-cells (leucocytes); detachment of the membrane of Descemet; disappearance of Bowman's membrane; new-formed tissue in the anterior chamber, and the corpus vitrei, the fibrillæ being well marked; thickening of the walls of the arteries in the ciliary body and choroid; atrophic degeneration of the retina."

This is a typical picture of glaucoma, lacking the feature of high intra-ocular tension. It could not be examined ophthalmoscopically because of the corneal opacity, and was designated *scleritis* from the most prominent clinical symptom of active localized disturbance.

Thus, instead of being a consequence of increased tension, the process which it shows to be in action is the probable cause of high tension, and all the other symptoms for which high tension has been held responsible. By reflex action through the vaso-motor center in the medulla oblongata, either from the encroachment of new connective tissue upon the ciliary and other nerves of the bulb, or by the excitement of these same nerves by an irritant contained in the blood; or by the direct influence of this irritant itself upon the vaso-motor

[29] *Archiv. Ophthal.*, Vol. XXIII., No. 4, p. 416.

center, causing contraction of the arterioles and increased heart's action (palpitation) to overcome the added resistance to the onward motion of the blood-stream, with consequent high arterial pressure and venous stasis, followed by dilatation of the arterioles in reaction, and the symptoms of inflammation, is the picture presented by irritative and inflammatory glaucoma, with increased intra-ocular tension, often relieving itself without interference, though not perhaps until visual power is abolished. A continued high tension is due to persisting venous stasis, as when tension *slowly* falls to the normal, after operation. If high tension depended upon the occlusion of the channels of filtration, nothing but mechanical interference would relieve it, when once "the vicious circle" had been formed; but we know that *passing attacks* of high tension are frequent. Eserin is effective in emptying the veins by the *spasmodic* pressure exerted by the iris muscle in its efforts to contract the pupil, and hence the greater efficiency of the weaker solutions, as a strong solution of eserin causes *rigid* contraction* of the iris.

High intra-ocular tension, due to the increased amount of blood in the vessels of the bulb, explains those cases in which the sinus of the anterior chamber is open, the variations of tension so common in *irritable* glaucoma, the advancement of the lens and iris, and the shallow anterior chamber; it accounts for a *hardness* of the globe which may at least balance the *vis a tergo*, the general blood pressure.

"When connective tissue is being supplied, the part becomes inflamed and swollen, owing to the exudation of plasma. The blood vessels become dilated and congested, and, notwithstanding the slower circulation, the *amount* of blood (in the part) is greater. The blood vessels are increased owing to the formation of new ones. Colorless blood corpuscles pass out of the vessels and reproduce themselves, and many of them undergo fatty degeneration, while others take up nutriment and become converted into large uninucleated protoplasma cells, from which giant cells are produced."[30]

Vascular disturbance is essential, but however this may be, connective tissue germination is wont to be accompanied by

* Perhaps well-devised manipulation of the eyeball would relieve tension. I have not tried it.

[30] *Foster's Text-book on Physiology*, 2d Ed., p. 405.

edema, and would be a more natural explanation of the edema of the cornea described by Fuchs.

Atrophy of the choroid, appearing earliest about the optic nerve in which an extra growth of connective tissue is so generally found, gives some color to Mauthner's[31] view:

"That glaucoma is a serous choroiditis by which vision is destroyed, and not by high intra-ocular tension; as the worst cases are those in which tension never rises above the normal."

The observation of Straub,[32] that when a meridional section of a glaucomatous eye is made, the choroid is found to have lost its elasticity, and does not withdraw from the sclera as in the healthy eye, is offered in evidence of its sclerosis, and its atrophy is due, at least in part, to this condition. Ulrich[33] regards sclerosis of the iris and infiltration as the two great factors in the pathogenesis of glaucoma, and Heyne[34] found in chronic glaucoma hyaline degeneration of the vessels. Valude[35] reported observations in four cases of hemorrhagic glaucoma, *all* of which gave evidence of arterio-sclerosis. The retinal vessels showed hyaline degeneration and peri-vasculitis. Optic nerve cupping, and closure of the iris angle were not always found, but dilatation of the iris and ciliary bodies was constant. He thinks this form of glaucoma due to an alteration of the retinal blood vessels, secondary to a disease of the general vascular system. Garnier[36] judges the compensatory endarteritis of glaucoma to begin in degenerated hyaline, or connective tissue masses between the elastic membranes. Schnabel[37] did an iridectomy on two eyes. There was nothing remarkable in the left; but from the right eye, with and behind the iris, a grayish mass prolapsed, which proved to be "a thin whitish membrane, pervaded by delicate straight and somewhat tortuous connnective tissue fibrillæ, and containing a few elastic fibers, numerous larger and smaller blood vessels filled with red blood discs; around these a larger quan-

[31] *Wien. Med. Bl.*, 10, p. 300.
[32] Report of Seventh International Ophth. Congress, Heidelberg, 1888.
[33] *Trans. Ophthal. Society*, Heidelberg, 1884.
[34] Inaugural Dissert., Königsburg, 1884.
[35] *Trans. Ophthal. Society*, Heidelberg, 1892.
[36] *Archiv. f. Augenheilk.*, Vol. XXV.
[37] *Archiv. Ophthal.*, Vol. VII., p. 277.

tity of fibrillar connective tissue, in which numerous cells were imbedded. * * * The exsected iris was thinner than normal." Prof. Jaeger had a similar experience in three iridectomies, and Dr. Kerzendorfer, in one. Schnabel[38] also reports sixteen, or more, cases of glaucoma, in all of which he found structure changes of the cornea (a cicatrix, an ulceration, or a staphyloma), or sclera, and closure or obstruction of Schlemm's canal, though some of them did not show increased tension. In some instances an increase of connective tissue was found in the ciliary muscle; and atrophy of the ciliary body first observed by Brailey,[39] exists in the great majority of cases of simple glaucoma, sometimes even before the glaucoma is manifest.

While only a portion of the recorded evidence is here offered, it would seem sufficient to establish the claims of this and a preceding paper (The Disease Process, Glaucoma, *American Jour. Med. Sci.*, June, 1893) that the local affection is a fibrosis, or connective tissue hyperplasia which, by the growth of the degraded tissue chokes to death the special functional tissues. The testimony of the eminent observers favors the further contention of this paper that the corneal appearance is *not symptomatic* of high tension, but is an organic change, shared in common with the other parts of the eye, especially with the sclerotic, whose density and opacity deny to us the observation of such slight manifestations as may be detected in the normally transparent cornea.

Repeating that it is the *type* of the disease, primary simple glaucoma, the basis of the varieties, which is under consideration, and recognizing that the evidence may not be conclusive to some minds, perhaps, because it has been lamely presented; or, because, from a long acceptance of the idea that high intraocular tension and glaucoma are almost synonymous, mental adaptability to any other view is impossible, the paper proceeds to the question:

What are the factors which most probably promote connective tissue hypertrophy?

Some of them have been named in the preceding pages.

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[38] *Archiv. Ophthal.*, Vol. VII., pp. 24 to 33 and 249 to 257.

[39] *Royal Lond. Ophth. Hosp. Rep.*, Vol. IX, 2, p. 199.

The chief examples of overgrowth of connective tissue in organs of high functional character are interstitial hepatitis, interstitial nephritis and sclerosis of the spinal cord.

Interstitial hepatitis may begin in a primary degeneration process, or in an irritative congestion. In either case cell degeneration seems to be the process, *and is often preceded by pain and enlargement for three or four years before the establishment of the sclerosis.* It may be secondary to malarial(?) hyperemia; due to abuse of alcohol; to diffusion of degenerative processes (as others coexist) which cause hypertrophy first, and are not inflammatory (Handfield Jones). It has been seen as early as 10 years of age (Frerichs). The morbid process involves extravasations of blood, complete destruction of the secreting structures, and disintegration and *partial absorption* of the component tissues of the organ.

Interstitial nephritis was shown in my paper, "The Disease Process, Glaucoma," to bear a close resemblance to glaucoma in its clinical history; in anatomical features it is of the same type. The *capsule* is less *transparent* than normal, with small vessels ramifying on its surface. In laying open the organ, the cut surfaces become convex, showing compression of its elements (tension). If the process is recent, the tissues are friable; if connective tissue overgrowth has taken place, the tissue is tough. There may be an accumulation of small round cells, and multitudes of new cells lying without their capsules.

Semmola[40] claims that true interstitial nephritis always consists in a general nutritive disorder, to which nephritis is secondary; beginning in diminished cutaneous respiration, followed by the imperfect digestion and transformation of albuminous foods. Palpitation of the heart, without organic change, conjoined with atonic or irritative dyspepsia is often found; observed in children as a heredity. A. Weber[41] found heart disease in all young persons having glaucoma, and that iridectomy served little purpose in such cases.

Sclerosis of the cord is a process of degeneration, though it may develop with some rapidity with symptoms of second-

[40] *Gazette Medicale de Paris*, 1875.

[41] *Graefe's Archiv.*, Vol. XXIII., No. 1.

ary degeneration; it is a primary irritation, with consecutive connective tissue proliferation (increase of neuroglia), and absorption of nerve fiber. *Causes:* Inherited tendency, constitutional syphilis, possibly sexual excesses, chronic alcoholism, repeated over-exertion and exposure to cold, and lead poisoning.

Thus, according to the most trustworthy views, connective tissue overgrowth is due to *dirty* blood; made so by imperfect digestion[42] (to which the habitual use of alcohol contributes), so-called struma, syphilis,[43] lithemia and lead poisoning; by the retention of effete matter and partially metamorphosed ingesta, through the inefficient action of the excretory organs. And glaucoma is found conjoined with albuminuria, eczema, palpitation of the heart, general high arterial tension, and constitutional syphilis; in fact with all the conditions which are supposed to foster the propagation of retrogressive tissue in organs of high functional value. Observation shows that excretion by the skin is very imperfect; that most of those who suffer from glaucoma have been accustomed to take cold still baths, thus increasing the contraction of the superficial arteries, and without the exercise which restores the circulation; and they eat too much for the waste they effect, and are habitually constipated.

It is claimed that high arterial tension is never absent in glaucoma, is always found in vaso-renal changes; is due to a chronic irritant of low intensity, in action through a considerable period of time; that it is the expression of Nature's effort to rid the circulation of the irritating element, and that it is always accompanied, or followed, by connective tissue hyperplasia.

The term *glaucoma* has had no real significance since the invention of the ophthalmoscope, and conveys to our minds no idea of the disease; hence, the substitution of the term *interstitial ophthalmitis*, descriptive of the pathologic changes common to all the structures of the globe.

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[42] "There is hardly any condition which is more certain to produce intense uric-acid-edemia than gastric catarrh." (Haig.)

[43] Within the past three years, I have seen two cases of simple glaucoma, clearly due to constitutional syphilis.

PROGNOSTIC SIGNIFICANCE OF ALBUMINURIC RETINITIS.*

By E. Oliver Belt, M. D.,
OF WASHINGTON, D. C.

PROFESSOR OF OPHTHALMOLOGY AND OTOLOGY HOWARD UNIVERSITY.
OPHTHALMIC AND AURAL SURGEON TO FREEDMAN'S HOSPITAL.

IT seems to be generally admitted that the retinitis, following scarlet fever and pregnancy, is not of special prognostic significance as to vision or mortality, for under treatment recovery of both vision and health are not unusual in these cases. Therefore in this paper such cases have been excluded as the prognosis is altogether different and unfavorable in the retinitis of chronic Bright's disease.

Though improvement of vision very frequently occurs in albuminuric retinitis, it is of minor importance in comparison with the life of the patient and the main point which I wish to determine is the average duration of life after the appearance of retinitis from renal disease. The statistics heretofore published upon this subject deal mostly with hospital cases in which the duration of life rarely exceeds one year. Occasionally we see reported a case in private practice which has survived five, ten and even fifteen years. (Noyes mentions one of ten years, and Webster reports the case of a minister who survived seventeen years.) This led me to think that we might be able to give a more hopeful prognosis in cases which could receive good medical attention with the proper hygienic

* Read before the Section of Ophthalmology, American Medical Association, Baltimore, Md., May 8, 1895.

surroundings, etc. And I decided to try and find whether or not the average duration of life was greater among private patients than among hospital cases. With this end in view a few weeks ago I requested quite a number of Ophthalmologists throughout the country to report the cases which had occurred in their private practice to me. The time allowed was too short for many to look over their records, and many had not kept trace of the patients, after referring them back to the family physician, but enough cases were reported to show that the duration of life is much longer among private patients, and that the renal affection is undoubtedly influenced by the hygienic surroundings. However, the number of cases surviving two years was disappointingly low, and the consensus of opinion, as shown by the following extracts from letters received, seems to be that nearly all prove fatal in less than two years.

Dr. G. E. de Schweinitz, of Philadelphia, says, "So far as I know there is no case in my private practice that has lived longer than two years after the development of the retinitis."

Dr. J. L. Thompson, of Indianapolis, says, "Have had between fifty and one hundred cases scattered through my books in a private practice of twenty years. Several of my patients have died within thirty days, while others have lived two years. Many more die within six months after its manifestation in the eye than live after that period."

Dr. Walter B. Johnson, Patterson, N. J., says, "In many cases the retinitis was the first indication of kidney disease, which, after discovery, almost invariably progressed very rapidly."

Dr. Peter D. Keyser, Philadelphia, says, "That the length of life depends upon the retinal appearance at the time of first examination. In well marked cases I should say an average of twelve months, if the patient is under good medical care. I have had some cases to run two years, others only three months."

Dr. L. A. W. Alleman, Brooklyn, N. Y., says, "Save in cases due to pregnancy, I have never seen a pure case of albuminuric retinitis last over a few months from the time it has come under my observation."

Dr. Samuel D. Risley, Philadelphia, says, "My impression is that after the appearance of eye symptoms nephritis is rapidly fatal. I recall cases of death occurring within a few weeks, and cannot recall one which did not prove fatal inside of two years, whether associated with pregnancy or not."

Dr. G. C. Savage, Nashville, Tenn., says, "My recollection of these cases is that they die within two months, and I do not remember a single case that lived longer than five months."

Dr. Charles W. Kollock, Charleston, S. C., says, "In no case in my practice has any such patient lived out the year."

Dr. Peter A. Callan, New York, says, "My impression is that the great majority die under two years after the onset of the retinal changes, but there are exceptional cases, one of which has now lived nine years since I first made out that she had the kidney trouble with the eye complication."

Dr. George T. Stevens, New York, says, "I observe that some of our colleagues think that a fatal termination is to be expected within a few months after the discovery of the albuminuric retinitis. I can recall to mind quite a number of people who have survived several years. Mrs. K. survived more than eleven years after I found well marked retinitis albuminurica. This is not, I think, a specially exceptional case."

Dr. David Coggin, Salem, Mass., says, "Of thirty fatal cases I remember but two who lived over six months."

Dr. C. S. Turnbull, Philadelphia, says, "My experience has, in a general way, led me to think that the time is most variable, depending greatly upon the many and varied forms of renal disease in different individuals."

From all the statistics I have been able to find we get the following results: Cases in private practice 155, of these 62% died within one year; 85% in two years, and 14% lived more than two years.

Hospital cases 77, of these 85% died within one year, 93% within two years, and 6% lived more than two years.

Mixed cases 187, of these 65% died within one year, 93% within two years, and 6% lived more than two years.

Total number of cases 419, of these 72% died within one year, 90% within 2 years, and 9% lived longer than two years.

	Died Within Time Designated Below.							
Physician.	Total Cases.	3 Mos.	6 Mos.	12 Mos.	18 Mos.	2 Years.	Over 2 Years.	Living after 1st observation.
Dr. J. H. Thompson, Kansas City	15			13				1—7 months 1—2 months
Dr. W. V. Marmion, Washington, D. C	10				10			
Dr. Hiram Woods, Baltimore, Md	4	1						1—6 months 1—5 months
Dr. William Cheatham, Louisville, Ky	9	1	2	2	2	2		1—18 months
Dr. George T. Stevens, New York	1						1—11 years	
Dr. S. C. Ayres, Cincinnati	2	1	1					
Dr. G. H. Good, Cincinnati	1					1		
Dr. J. A. Spalding, Portland, Me	6	1	2	1		2		
Dr. H. B. Young, Burlington, Ia	3	2					1—5 years	
Dr. David Coggin, Salem, Mass	30		28				2	
Dr. Richmond Lennox, Brooklyn, N. Y.	2	1	1					
Dr. P. A. Callan, New York	1						1—9 years	
Dr. W. F. Mittendorf, New York	6	3	1		1	1		
Dr. E. C. Rivers, Denver, Col	3			2				1—8 months
Dr. R. J. McKay, Wilmington, Del	11		5	3	2		1—3½ years	
Dr. E. Oliver Belt, Washington, D. C	2		2					
Total	106	10	42	21	15	6	6	6
Total	Deducting the 100	six cases 10% Died	not traced two 42% within Died	years 21% one year within	leaves 15% 73%. 2 years	leaves 6% 94%.	the following: 6%	

				Died	within	2 years	94%.	

		Died Within Time Designated Below.						
Physician.	Total No. of Private Cases.	3 Mos.	6 Mos.	12 Mos.	18 Mos.	2 Years.	Over 2 Years.	Living after 1st observation.
Dr. G. Hartridge	5	1	1	3				
Dr. James Anderson	3	2	1					
Dr. Simeon Snell	8	3	3	1	1		4—3 years	
Drs. Possaner & Haab	39			19		4	2—8 years	10 two to five years
							10—2 to 5 years	
Dr. E. Oliver Belt	100	10	42	21	15	6	6	
Total	155	16	47	44	16	10	22	
	Died	within	one year	62%				
		Died	within	two	years	85%	14%	
	Hospital Cases							
Dr. Miles Miley	45	26	11	5	3			
Drs. Possaner & Haab	33			24		3	5	3 over two years. 1 less
Total	78	26	11	29	3	3	5	
	77—	1 case	not traced two		years.			
	Died	within	one year	85%				
		Died	within	two	years	93%	6%	
	Mixed Cases.							
Dr. C. S. Bull	103	16	14	27	7	11	6—3 years	
							4—4 years	14 six months
							1—6 years	2 one year
							1—7 years	
	Deducting 16	casesnot	traced 2	years	leaves:			
	87	16	14	27	7	11	12	
Dr. Gruening	100					100		
Total	187	16	14	27	7	111	12	
	Died	within	one year	65%				
		Died	within	two	years	93%	6%	
Private	155	16	47	44	16	10	22	
Hospital	77	26	11	29	3	3	5	
Mixed	187	16	14	27	7	111	12	
Total	419	58	72	100	26	124	39	
	Died	within	one year	72%				
		Died	within	two	years	90%	9%	

THE RECONSTRUCTION OF THE LID BORDER IN ENTROPIUM OF THE UPPER LID.[1]

By F. C. Hotz, M. D.,
OF CHICAGO.

THE correct position of the eyelashes is so thoroughly dependent on the presence and correct position of the free border that no operation which fails to reconstruct the lid border, can successfully and *permanently* remove the entropium.

As long as the tarsus possesses its normal elasticity the inverted border can be turned back to its normal position by the operation I introduced in 1879.[2] But in the higher degrees of entropium the tarsal cartilage has suffered such structural changes that it has lost its elasticity; and its rigidity, then, is a serious obstacle to the reposition of the free border. Under these circumstances the reposition is possible only if a wedge-shaped piece is removed from the cartilage just above the line of the eyelashes, the excision being made in such a manner that the apex of the wedge-like wound is located near the mouth of the Meibomian glands.[3]

This grooving operation, however, can be carried out successfully only if the tarsus is large and thick enough to allow the removal of a wedge of the requisite size, but in the worst forms of entropium the cartilage is often shrunken to a small and thin plate which offers a very poor chance for the

[1] Clinical lecture at the Chicago Policlinic, May 29, 1895.

[2] See *Archiv. f. Ophthal.*, Vol. viii, p. 249.

[3] See my paper read before the Ninth International Medical Congress, 1887.

proper grooving operation. In such cases skin grafting can help us over the difficulties; for if we cannot turn back the whole inverted lid border we may turn up its anterior edge containing all the eyelashes and support them in their correct position by the implantation of a strip of skin forming a new or artificial lid border.

The first attempt at creating an artificial lid border is found in Spencer Watson's operation 1873; from this crude beginning the operation has gradually been improved by a process of evolution in the various transplantations suggested by Gayet, Dianoux, Jacobson, and many others, and has finally reached its present simple and perfect form by the use of skin grafts.

I perform the operation in the following manner: The lid border is split by the well-known intermarginal incision, which is made so deep that the anterior edge of the lid can be turned up with perfect ease. Now I make a transverse incision through the lid skin and orbicularis muscle just below and parallel with the upper line of the tarsal cartilage, excise the strip of muscular fibers which cover the upper border of the cartilage, and unite the lid skin with the upper border of the cartilage by three sutures. One suture is placed at the center of the wound, and one at either side of the central one. Each suture passes through the edge of the lid skin, then through the upper border of the cartilage and finally through the upper edge of the skin wound. When these sutures are tied the lid skin is drawn upwards and fastened to the upper border of the tarsus. This traction upon the lid skin is sufficient to cause a thorough eversion of the anterior edge of the split lid border, and when the anterior edge is thus turned up and separated from the posterior edge the intermarginal incision becomes a gaping wound several millimeters in depth and with sloping sides. This groove is to be filled by a skin graft, which must be thicker than a Thiersch shaving, long, narrow and somewhat wedge-shaped. Sometimes I have used for such grafts a narrow strip trimmed off by scissors from the upper or lower skin border of the wound in the lid, but I prefer taking them from the skin behind the ear, where the skin is of a firmer texture and where the presence of the smooth bone

surface to which the skin is attached is of great assistance in the accurate cutting of a long and slender strip; for as this strip is to be but from $1^1/_2$ to 2 *mm.* in width, it is very essential that the skin does not drag before the knife or scissors (as the loose lid skin is apt to do) because this would make the width of the graft so uneven as to render it unfit for use. I first make a longitudinal incision about 1 *mm.* deep and as long as the graft is to be. Then a second incision is made parallel to the first one and at a distance of $1^1/_2$ or 2 *mm.* according to the width of the intermarginal groove the graft is intended to fill. This second incision is made to join the first one at both ends and to meet it at a depth of 1 *mm.* by giving it a slanting direction. The narrow wedge-shaped strip of skin thus mapped out is seized by a fine forceps, completely dissected off and transferred directly to the wound in the lid border which previously has been carefully freed from all blood coagula. The graft is spread out and gently pressed into the groove, and if it fits nicely the lid is once more irrigated with the salt solution. Both eyes then are covered with compresses and bandage in order to stop all movements of the lids. After twenty-four hours the graft is so firmly adherent that the bandage may be left off. Should the graft be too long or too wide the ends or edges can be trimmed down with scissors without taking the graft off from its new resting place. But if it is too thick and stands out beyond the plane of the wound edges I turn it over with its epidermis surface down, shave off a little of the cutis with fine scissors and put it back in place again.

During the first two weeks the epidermis of the graft is repeatedly shed; it is, therefore, advisable to keep the new lid border well lubricated with vaseline or a simple ointment lest the dry and hard epidermis scales annoy and irritate the eye. After this period of desquamation the surface becomes smooth and the engrafted piece can hardly be distinguished from the rest of the lid border.

I regard these skin grafts a much better material for the reconstruction of the lid border than the grafts of the Thiersch kind or of mucous membrane, for we have to deal with a groove-like wound which has a great tendency of

closing up from the bottom and drawing its edges together. The implantation of a solid graft will resist this contraction far better than if we only line the surface of the wound with epidermis or mucous membrane.

The use of such skin grafts has been objected to on the ground that they might contain fine hairs which would prove a new source of irritation to the eye. Judging by my own experience I must pronounce this fear groundless. I have never seen any hairs grow in these grafts and I am inclined to the belief that when any hairs are found, a careful examination will show that they do not come out of the graft, but from the posterior edge of the lid border; in other words they are eyelashes which were left in the posterior edge when the lid border was split.

HEREDITARY RETRO-BULBAR NEURITIS.

By Oscar Dodd, M. D.,

ASSISTANT SURGEON ILLINOIS CHARITABLE EYE AND EAR INFIRMARY; CLINICAL INSTRUCTOR, OF EYE AND EAR, COLLEGE OF PHYSICIANS AND SURGEONS, CHICAGO.

HEREDITARY amaurosis was known to exist in pre-ophthalmoscopic times, but owing to the absence of precise methods of diagnosis it was confounded with retinitis pigmentosa and other diseases of the fundus. Graefe,[1] in 1858, described a case of retro-bulbar neuritis, being the first recorded in which an ophthalmoscopic examination had been made. In 1865 he described other cases, but it remained for Leber,[2] in 1871, to give a graphic description of the disease. He there reports fifteen cases of his own, in four families, and gathers the records of the authentic cases previously reported. Other cases have been added to the literature by Fuchs,[3] Norris,[4] Keersmaecker,[5] Habershon[6] and others, but practically nothing has been added to description of the disease as given by Leber. Having had an opportunity to watch a case of this kind for several months I thought it of interest to report it and call attention to some of the peculiar characteristics of the disease:

Myrtle E., 14 years of age, came to the clinic of the Illinois Charitable Eye and Ear Infirmary, March 30, 1894, and gave the following history: About two months ago she began to have severe frontal headaches two or three times a week which lasted for a day or more. About a month later she noticed that the sight of her right eye was failing, and a few days after that the left began

to grow dim. Her eyes were normal in appearance and the ophthalmoscope showed only a slight congestion of the vessels in the right eye and a faint pallor of the temporal part of the optic disc in the left eye. There was also a large, deep physiological cupping of the disc which made it more marked. Her vision in the right eye was $\frac{12}{200}$, and with the left she could count fingers at 1 foot. Her fields of vision as taken at this time showed some contraction and a poor perception for colors, but no scotoma could be defined. Her general health had always been good and there was no appearance of any hereditary or constitutional disease.

Her family history revealed the fact that a sister had an eye trouble similar to hers, beginning at 12 years of age, with severe headaches and progressing blindness, which remained constant for several months and then improved to some extent. It continued in this condition until her death, caused by an accident, at 34 years of age. The history did not reveal any eye trouble of this nature in the family previous to these cases, but their knowledge of the other members of the family was very meager. The father is living and has never had any eye trouble. He has been addicted to excessive alcoholism all his life, as well as his father before him. The mother died at the age of 54 from pneumonia. She was myopic, but otherwise had no eye trouble. Three sisters died in infancy. One sister is living, healthy, and never had any eye trouble.

The patient was given strychnia, $\frac{1}{30}$ grain, three times a day for a month, when her vision was: R. E. $= \frac{12}{200}$; L. E. $= \frac{8}{200}$. The discs had become paler, especially on the temporal side. Hypodermatic injections of antipyrin (7 to 14 grains) every second day, as recommended by Valude, were then given, with no result, but a more normal coloring of the disc and a gradual failing of vision. May 4, R. E. V. = fingers at 6 feet; L. E. V. = fingers at 4 feet. The vision was very inconstant, varying with the position of the eye, as central vision was completely absent. An attempt was made to map out the scotoma, but she could not fix the eyes sufficiently to do so. On September 21, vision equalled fingers at 5 feet with each eye. There is a large positive central scotoma which can be easily defined and she has developed the faculty of eccentric fixation so she can use the eyes better than formerly.

She disappeared from the clinic and I did not see her again until she came to my clinic at the College of Physicians and Surgeons the last of November. The central scotoma had grown larger and the discs were much paler. The whole nerve now appears atrophic, but the atrophy is more marked on the temporal side. V. = fingers

at 3 feet with each eye. In January I again examined her and found the fields of vision and sight nearly the same as in November.

The characteristics of the disease are very similar to those of toxic retrobulbar neuritis at the onset. It differs from it, however, in the ultimate destruction of that part of the optic nerve supplying the central region. The disease is usually preceded by severe frontal headaches and pain back of the eyes. The destruction of central vision progresses quite rapidly, so that in a few weeks it is limited to the perception of large objects with the peripheral part of the retina.

The age at which it most frequently occurs is about that of puberty, although some cases are reported which occurred as early as 8 years and others as late as 45. In males it almost always occurs between 15 and 25 years, while in females there seems to be two periods of frequency — puberty and at the menopause. It is much less frequent in women than in men. According to De Wecker, the proportion is 8 or 10 to 100, but among the authentic cases in the literature I found 13 females to 39 males. Leber[7] believes that when it occurs in women it takes a much more malignant course.

The transmission of the disease is rarely direct, but is usually through a healthy female to the second generation. It is easily traced in some families through several generations, while in others there is only the fact of its occurrence in several members of the same family at about the same age. The disease usually follows a similar course in all members of a family, but varies much in course and severity between different families. The occurrence of syphilis or excessive alcoholism in the parents is said by De Wecker to have no demonstrable effect in its production. This, however, seems to me to be very questionable, as we know the tendency of the children of such parents to other diseases of the nervous system. In the case reported we have no other cause apparent except the tendency to the disease transmitted by the father and grandfather, who were inebriates most of their lives.

The analogy between toxic and hereditary retro-bulbar neuritis is very great. In many cases of toxic retro-bulbar neuritis there must be a tendency to the disease or weakness

of that part predisposing it to an attack, as the amount of the toxic substance taken is not large. So we may consider the tendency so slight in these cases as to require some toxic influence to produce the disease, while in the hereditary form the tendency is so great as to require only ordinary conditions to produce it, as the changes at puberty, so that it manifests itself in several members of a family or even in several generations, as an "idiopathic" disease.

The diagnosis of the disease can only be made from a careful history of the patient's family, as it presents no distinct symptoms which may not be found in other diseases of the optic nerve. It is most likely to be mistaken for toxic retro-bulbar neuritis, as it presents a very similar appearance at an early stage. Its rapid progress, the destruction of the central fibers of the nerve and the supervening palor and atrophy of the temporal side of the disc when combined with the history of a similar trouble in other members of the family will be enough to fix the diagnosis of hereditary retro-bulbar neuritis.

The prognosis of the disease is bad as far as any hope of improvement of vision from treatment is concerned. It is favorable, however, in so far that complete blindness does not take place. The vision may not return sufficiently for any particular work, but by the development of the peripheral part of the retina it may improve to some extent. Norris records two cases where the vision, after remaining very poor for some time, increased to $^{6}/_{30}$, and enabled them to do ordinary work.

Treatment is apparently of no benefit in most cases, as the disease steadily progresses in spite of it. Despagnet thinks, however, that those cases in which it is carried out thorougly do not develop such a degree of blindness as those in which it is not used. This is, however, hard to determine, for the extent to which it progresses varies much among different families.

The points to which I would call particular attention in this disease are:

Its resemblance at its beginning to the ordinary cases of toxic amblyopia of a severe type.

The rapid destruction of vision in the central part of the retina combined with very slight changes in the optic disc.

The variableness of the vision due to the inability to use the peripheral portion of the retina.

The ineffectiveness of treatment to influence the course of the disease.

The fact that total blindness does not result, although the vision remaining may be of a low degree.

REFERENCES.

[1] Von Graefe: "Ein ungewöhnlicher Fall von hered. Amaurose," *Graefe's Archiv.*, 1858.

Sedgwick: "A Case of Hereditary Amaurosis," *Med. Times and Gazette*, 1862.

Von Graefe, *Zehender's klin. Monatsblatter*, 1865.

Mooren, *Ophthalmische Beobactungen*, Berlin, 1867.

[2] Leber: "Ueber hereditäre und congenital angelegte Sehnervenleiden," *Graefe's Archiv.*, 1871.

Hutchinson, *Ophthalmic Hospital Reports*, Vol. VII., 1871.

Daguenet et Galezowski: "Variété non encore decrite d'amaurosis congenitale," *Journal d'Ophthalmologie*, 1872.

Prouff: "Sur une forme d'atrophie papillaire observée chez plusiers members d'une même famille," *These*, Paris, 1873.

Mooren, *Ophthal. Mittheilungen*, Berlin, 1874.

Alexander: "Drei Fälle von hereditäre Sehnervenleiden," *Zehender's klin. Monatsblatter*, 1874.

Pufahl: "Ueber hereditäre Amblyopie," *Berliner klin. Woch.*, 1876.

[3] Fuchs, *Zehender's klin. Monatsblatter*, 1879.

Galezowski: "Des amblyopies et des amauroses congenitales," *Rec. d'Ophthal.*, 1879.

Higgins, *Medical Times and Gazette*, 1880, and *The Lancet*, 1881.

[4] Norris: "On Hereditary Optic Atrophy," *Trans. of the Am. Ophthal. Soc.*, 1880-1884.

[5] Keersmaecker: "De l'atrophie axiale du nerf optique observée chez plusiers members d'une même famille," *Rec. d'Ophthal.*, 1883.

Story: "On Hereditary Optic Atrophy," *Trans. of the Academy of Medicine of Ireland*, 1884.

[6] Habershon: "Hereditary Optic Atrophy," *Trans. of the Ophthal. Soc. of the United Kingdom*, Vol. VIII., 1887.

[7] Leber, *Graefe und Saemisch Handbuch f. Augenheilkunde*, Vol. V., 1877.

THE OBLIQUE MUSCLES AS RELATED TO OBLIQUE ASTIGMATISM:

REPLY TO DR. HOTZ'S CRITICISM.[1]

By G. C. Savage, M. D.,
of Nashville, Tenn.

Editor of Annals:

I plead guilty to the charge of having "talked" much and "written" more on this subject. I plead guilty, further, to the charge of having taught one thing (harmonious nonsymmetric action) in 1887, and another thing (harmonious symmetric action) in 1891. To have taught an error is to no man's credit. It was my good fortune, however, to detect this error. I quickly exposed the incorrectness of my 1887 teaching—just as quickly as if another had been the unfortunate one. The main thought of my first paper was that oblique astigmatism was more annoying than the vertical or horizontal, because, in the former, the oblique muscles were involved. This was no error. I then knew nothing of the obliquity of images in oblique astigmatism; and, believing the old teaching that the oblique muscles must always keep the naturally vertical meridians parallel, I readily fell into the error of teaching that the action of the obliques, caused by oblique astigmatism, was "harmonious nonsymmetric." I at that time conceived the purpose of the rotation to be to bring the meridian of best curvature to, or as far as possible toward, the vertical or horizontal position, a work in which the obliques were often aided by a leaning of the head toward the shoulder.

[1] On the alleged action of the oblique muscles in oblique astigmatism, Annals of Ophthalmology and Otology, Vol. IV., No. 2, April, 1895.

I then thought the vertical (and horizontal) astigmatism gave least trouble because of the mere fact of position; but now I can understand why oblique astigmatism with meridians of greatest curvature parallel, will give as little trouble, for in each of these conditions any object will throw similar images on corresponding retinal parts. For the reason that the obliques do not have to perform their complicated function in these forms of astigmatism, their correction is never attended by metamorphopsia or other annoyance.

I try always to "have a reason for the faith that is within me," and any change of faith or teaching on my part must be based on reason. My reason for abandoning the teaching of harmonious non-symmetric action of the oblique muscles in oblique astigmatism, and, in its place, teaching that these muscles must act symmetrically, is that the refraction of such

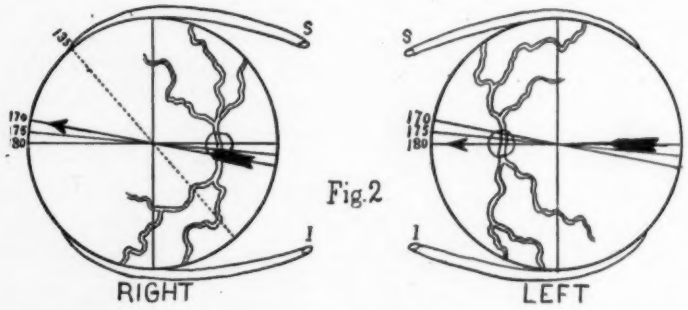


Fig. 2

eyes cause the formation of dissimilar images on non-corresponding parts of the retinæ. This thought of oblique images in oblique astigmatism occurred to me one night early in 1891, and the next day I was able to demonstrate its correctness, not only to my own satisfaction but also to the complete satisfaction of Dr. G. H. Price. I did not then remember that Dr. J. A. Lippincott had taught us this in the *Archives of Ophthalmology*, April, 1889.

Dr. Hotz complains that I have not given my "method of observation," and that I failed to set forth "the tests or experiments" that led me to my conclusions, so that others might judge for themselves as to the correctness of same. This accusation was certainly inadvertent, for a little further

on (page 105 of your last issue) he quotes my experiment in part. I give it here in full: "The obliquity of the images in oblique astigmatism is a matter demonstrable. One can artificially produce any kind of astigmatism. One who is emmetropic, or at least is non-astigmatic, by placing a — 3 D. cyl. before each eye in trial frames, creates 3 diopters of hypermetropic astigmatism. The axis of the left cylinder being at 90° and that of the right at 135°, he has made of his own eyes the kind represented by Fig. 2. He may now for a moment place the opaque disc in front of his right eye, at the same time placing the double prism (each 6°) before the left eye. A horizontal arrow, head to left, having been drawn on a card board, he looks through his double prism and sees two horizontal, hence parallel arrows. On removing the opaque disc from the right side of the trial frame a third arrow appears between the other two, but not parallel with them—it

FIG. 8.

is oblique down and to the patient's left. On removing the double prism two arrows are at once readily seen, the one crossing the other, as in Fig. 8. In a moment the two arrows begin to shut and open like the blades of a pair of scissors, and finally they are merged indefinitely into one."

I commenced the experiment, convinced that artificial astigmatism would produce the same image-changes which result from natural astigmatism, and what convinced me was that my 3 D. artificial hyperopic astigmatism was thoroughly corrected by a + 3. D. cyl., axis coinciding with the meridian of unaltered curvature. The above experiment seems to me to be convincing. I have other reasons for believing in the obliquity of images and will give some of them in words I recently used in THE JOURNAL. "Let Dr. Hotz take any case of astigmatism of more than 3 D. with the meridian of greatest curvature either vertical, horizontal or oblique, and, if the patient has ordinary intelligence, he can soon satisfy himself that astigmatism is not only capable of blurring, but also of distorting an object. One eye should be excluded while the

patient is asked to look at a rectangular card, two by four inches, held vertically immediately in front of the patient, at the reading distance. As the card is revolved on a pin piercing its center the patient should be asked its shape when at three definite points. When the long sides of the card are parallel with the meridian of greatest curvature, the patient will say it is a rectangle; when these sides form an angle of 45° with the meridian of greatest curvature, the answer will come quickly that it is a parallelogram; again, when these sides are brought to right angles with the meridian of greatest curvature, the card again is seen as a rectangle. But possibly Dr. Hotz is ready to say that these statements as to the distortion of the object prove nothing as to the distortion of the retinal image. Let us see: The law of projection (direction) is supreme in monocular vision, therefore, the lower border of the retinal image must be in the same plane with the upper border of the card, and so on for all the borders of the card and image, and these planes must all cut the nodal point. Then in obedience to this law the image must be distorted when the object appears to be.

"Only one other argument as to the distortion of the retinal image in an astigmatic eye, when the object is held so that its outlines are oblique to the principal meridians: all will agree that the meridian of least curvature is the line of union of the bases of the prismatic arrangement of the astigmatic cornea, and that all prisms refract light towards the base. Let us then take the right eye of a case of astigmatism with the meridian of least curvature at 135°; a horizontal line held before this eye will send its light from its entire length into the eye, but for convenience of study we will consider only the axial rays coming from the two extremities. The ray from the left end of the line strikes above the meridian of least curvature and must be bent towards it, its subsequent course in the eye necessarily being down and out; the axial ray from the right end of the line strikes below the meridian of least curvature and must be bent towards it, the course of this ray after refraction being up and in. Thus it is easily shown that the image of this horizontal line must be inclined down and to the right. Because of this inclination of the

image, the line itself seems inclined to the same extent and in the same direction. This is one law of physiologic optics.

"The distortion of retinal images in monocular vision is settled by the law of direction; the rotation of the eyes by the oblique muscles, in oblique astigmatism, is compelled by the more powerful law of corresponding retinal points."

Dr. Hotz made the following quotation from one of my papers: "In oblique astigmatism, be the obliquity much or little, it is a physical impossibility for the horizontal object and the retinal image to occupy the same plane. The same is true of all objects not in a plane with one or the other of the two principal meridians." Forgetting that the closing sentence of the above quotation had been made, the doctor informs me and your other readers that "the objects in nature are not all horizontal arrows, but present also vertical and oblique outlines," "unfortunately for Dr. Savage's theory." The fact that when oblique astigmatic eyes attempt to fuse images of a horizontal line, the images of a vertical line harmonize less, which Dr. Hotz thinks unfortunate for my theory, is favorable to my teaching. In an editorial in the *Ophthalmic Record*, referring to Dr. Wilson's criticism published in the *Archives of Ophthalmology*, I conceded that the same eyes could fuse images of either horizontal or vertical lines when they existed alone; for example, the meridians of greatest curvature diverging above, the superior obliques would cause the fusion of the images of a horizontal line, and the inferior obliques would fuse the images of a vertical line. In the same editorial I taught when both horizontal and vertical lines are viewed that the eyes attempt the fusion of the horizontal lines only; but was unable then, and am unable now, to give any reason for this. I only know it to be a fact. My knowledge of the fact rests on these observations which may be repeated by anyone: In a case of 3 D. astigmatism, with meridian of greatest curvature at 135° for right eye and 45° for left eye, if a rectangle be looked at by the right eye alone it will be seen as a parallelogram, leaning down and to the left; by the left eye alone, a parallelogram, leaning down and to the right; with the two eyes together it will be seen as a trapezoid with the longer side above. By action of the

superior obliques the upper borders of images (lower border of object) are completely fused, while at the same moment all of the lower border of right image, except its inner extremity, is fused with a corresponding portion of the same border of the left image. The parts of the lower borders of the two images not fused are directly continuous with the fused portion, hence the greater length of the upper border of the object. Of the two diverging borders, the right one is seen by the right eye, and the left one by the left eye.

If the meridians of greatest curvature had converged above the action of the inferior obliques, in binocular vision, would have converted the rectangle into a trapezoid with the longer side below. As already stated, this preference for fusing horizontal lines is distinctly shown in the higher degrees (3 D. or more) of natural oblique astigmatism. It is also distinctly shown in similar degrees of artificial astigmatism. The chief purpose of the existence of the recti and the *oblique* muscles is the fusion of images in binocular vision.

As to Dr. Hotz's experiment with his + 10 ⊃ + 2 cyl. I have this to say: His slit in the metal screen was entirely too short ("one inch") to be so far removed ("several feet") from the surface representing the cornea. Necessarily this slit, at such a distance, would throw a very short image on the ground glass four inches behind the lens, and thus make it very difficult to detect the slight leaning (less than 2°) of the borders, caused by the weak cylinder used. A slit several inches long and only a few feet away from the lens would have thrown a much longer image on the ground glass, so that the very slight change in direction of the image borders could have been easily detected. Dr. Hotz's report of this experiment is as strange as fiction though given as a fact: when the slit occupied the horizontal position, the cylinder, when revolved, inclined the vertical lines without altering the direction of the horizontal lines; when the slit was turned to the vertical position, the same cylinder, when revolved, inclined the horizontal lines without changing the direction of the vertical lines. A lifeless lens cannot show any partiality to rays of light coming from horizontal or vertical lines that bear the same relationship to the cylinder axis.

Now allow me to try to break in pieces the "key-stone" of his argumental arch, viz: the clinical test to which he put my theory. The case reported was one of mixed astigmatism, and the cylinder given the right eye probably was a — 3 D., and that for the left eye a — 2 D. The meridian of greatest curvature in O. D. was 115°; in O. S. at 65°. (These meridians diverged above. The image of a rectangle would have been distorted down and in by each eye, and in binocular vision these leaning parallelogram images would have fused so that the patient would have seen a trapezoid long side above.) The correcting cylinders were given and metamorphopsia was observed at once by the patient, but he does not give the character of the changed vision. Without the correcting lenses on he resorted to a test, the double prism, which he states I had advised, and he found all three lines parallel.

My first strike at this "key-stone" will be with the statement that I have never resorted to this test by the double prisms in uncorrected natural oblique astigmatism, nor have I ever advised it. I would expect the lines to be parallel, for the obliques, from habit, would so rotate the two eyes as to make the lines parallel. If he had used the double prism test on his patient while wearing her spectacles, and still troubled with the metamorphopsia, he would have found want of parallelism of the middle line with the two other lines; it would have inclined down and toward the corresponding side. The same test applied after the disappearance of the metamorphopsia, the spectacles being on, would show parallelism of the lines; but removing the lenses at this time the test would show the middle line leaning down toward the opposite side. The explanation of all this is easy:

The double prism test in oblique artificial astigmatism (this I did advise) will always show the dipping of the middle line, because the habit of rotation by the obliques has not been established. In Dr. Hotz's case of mixed oblique astigmatism the superior obliques had been always in the habit of rotating the eyes, and this habit reasserted itself when the eyes were under the double prism test, as may always be expected, and the lines were parallel. If the metamorphopsia has disappeared under the wearing of the lenses, the double prism test

applied to the naked eyes will show the middle line dipping down and toward the opposite side, for the habit of rotation has been broken. There never was (there never will be) a time in this case when, with the naked eyes, there was not metamorphopsia of that kind which would have made a rectangle appear as a trapezoid, the longer side above. To this form of metamorphopsia the patient had always been accustomed, and therefore was not worried about it. The form of metamorphopsia with the lenses, when they were first given, was a new kind—the trapezoid had its long side below, necessarily, though the doctor did not tell us so much. Being new to the patient, it was naturally annoying. An explanation of this new metamorphopsia is easy: while carrying the patient through the examination for the lenses, he, of course, excluded one eye. The uncovered eye naturally rolled into the position of rest for all the ocular muscles, and the axis of the cylinder for that eye was located. Similarly the cylinder was given the other eye. In binocular vision the old habit of rotation by the superior obliques was reasserted, and there was a consequent loss of coincidence of the axes of the cylinders and the meridians of least curvature (the cylinders were concave), the cylinder axes being thrown (about 3°) in the arcs of distortion for the inferior obliques. In this case the metamorphopsia could disappear only by the superior obliques giving up work which the inferior must take on. Without the lenses the superior obliques have been forced to converge the naturally vertical meridians; with the lenses on the inferior obliques must parallel these meridians. Usually this change is quickly accomplished in cases like this one. Dr. Hotz will not deny that the cylinders given changed the direction of the images of vertical and horizontal lines. To my mind it is clear that the leaning of images caused by the lenses was equal in extent, but opposite in direction, to that produced by the astigmatic cornea. The lenses have only rectangled the images of the rectangular figure. If in these cases the oblique muscles would only allow the cylinder axes and the meridians of best curvature to remain coincident in binocular vision, there would be no such thing as metamorphopsia ever complained of. It is never observed by patients

having astigmatism equal in the two eyes and the best meridians parallel, though they may be oblique. When the meridians of greatest curvature converge above, the use of correcting cylinders is always attended by metamorphopsia which is slower to disappear than in cases like Dr. Hotz's.

Dr. Hotz's closing paragraph, if true, would wipe out all that I have ever written about oblique astigmatism and the oblique muscles. This is his language: "It is, therefore, evident that neither experiments nor clinical observations nor the laws of physiological optics sustain the doctrine of the obliquity of the retinal images and the necessity of any action of the oblique muscles in oblique astigmatism."

In another part of this reply I have shown conclusively that both experiment and clinical observation prove my teaching to be correct, so that two of his three witnesses against my views have been made to testify in favor of them. It is even easier to capture his third witness (physiologic optics) and thus make the trio give evidence the very opposite to that which he intended they should give. Without doing violence to the laws of physiologic optics, I may state that every point of an astigmatic cornea has two radii of curvature, one the radius of spherical curvature, the other the radius of cylindrical curvature.* In the horizontal meridian of a vertical astigmatism, these two sets of radii are in the same plane, hence the rays of light entering the eye in the horizontal meridian, would be in the same plane after the refraction as before. Above or below the horizontal meridian, and out or in from the vertical meridian, there is not a corneal point that would give us these two radii in the same plane; the radius of spherical curvature would go to the center of the sphere while the radius of the cylindrical curvature would necessarily be in a horizontal plane. Both of these radii will be directed toward the plane of the vertical meridian, but they diverge as they go. A ray of light striking such a point must undergo a double refraction (a resultant refraction). If the corneal point is thus related to the horizontal and vertical meridians, the ray of light passing through it must be deflected

* There is only one radius, the resultant of the two. It is the radius, not of a spherical or cylindrical surface, but of a sphero-toric surface.

towards each of the two radii, therefore the refracted ray can no longer occupy any plane in common with the incident ray.

Allowing the retina to remain in its normal position, let us revolve the astigmatic cornea discussed above so that the astigmatism shall be oblique, at an angle of 45°. In doing this we have not altered the relationship of the two sets of radii—those that were in the same plane before, are so now; those that diverged before, diverge still. Let us conceive it to be the right cornea and that the meridian of least curvature now stands at 45°. The meridian that was at 45° when the astigmatism was vertical, stands at 180° when the astigmatism is oblique at 45°. What happens now to the axial rays in the horizontal plane? We will take three of these rays and follow them as they make their way back to the retina: one ray is from the middle of a horizontal line (arrow, if you please), one is from one end of the line and the other is from the other end. These rays must converge toward that part of the cornea in front of the pupillary space. They come to the eye in the same plane and strike the horizontal meridian of the cornea. The point of fixation is the center of the line, therefore the middle of these three rays strikes the center of the cornea vertically, and therefore coincides with both the radius of spherical and radius of cylindrical curvature for that point, hence passes through the cornea without being refracted at all and *impinges on the retina's horizontal meridian.* The ray from the right end of the line strikes the right side of the cornea at a point in the horizontal meridian, the two radii of which diverge, the radius of sperical curvature being in the horizontal plane, the radius of the cylindrical curvature pointing down as well as towards the plane of the meridian of least curvature. It is clear that this ray must undergo a double (resultant) refraction. Striking the cornea to the temporal side of radius of spherical curvature, it is so refracted as to converge less towards the middle axial ray (in this it is aided by the cylindrical curvature), and being incident to the radius of the cylindrical curvature on its lower side, it must be refracted down also. Following its subsequent course we find *it impinging on the retina below the horizontal meridian, to the nasal side of the vertical meridian.* The ray from the left

end of the line, coming to the cornea in a plane with the other two, strikes the cornea on the nasal side of its center and in the horizontal meridian, at a point from which the two radii diverge, the radius of spherical curvature being in the horizontal plane, the radius of cylindrical curvature pointing up and toward the plane of the meridian of least curvature. Striking the cornea on the nasal side of the radius of the sphere, this ray is made to converge less toward the middle ray than before refraction; striking the cornea on the upper side of the radius of cylindrical curvature, it is also refracted upwards. Following this ray to the retina we find *it impinging above the horizontal, and on the temporal side of the vertical*, meridian. A line drawn through these *three points of impingement* will locate the *image* of the line (arrow) looked at. It is inclined in obedience to the law of refraction that a ray of light in passing from a rarer into a denser medium must be refracted toward the perpendicular at the point of impingement.

There is one other objection which Dr. Hotz brought forward, viz: While a concave cylinder held obliquely in front of an eye at some distance will make horizontal and vertical lines appear inclined towards its axis, this inclination grows less and less as the eye is approached, and, as he thinks, disappears entirely when the cylinder is brought into contact with the cornea. This is all easily explained. Take again the three axial rays in a horizontal plane. Striking the horizontal portion of the oblique cylinder, the middle ray passes through unrefracted and continues in the same plane, while one of the outer rays is made to deviate downwards, and the other upwards. For convenience of study we will say that the deviation of each ray is 2° from the horizontal plane. This deviation continues the same until the retina is reached, regardless of whether this distance is 1 *m.* or 25 *mm.* In obedience to the law of direction the horizontal line is made to appear to incline more when the cylinder is held 1 *m.* from the eye than when it is held 50 *cm.* away. In obedience to the same law the line appears less and less inclined as the oblique cylinder is made to approach still nearer the eye, but even when brought into contact with the spherical cornea, its inclination does not and cannot disappear entirely, though often one

may not be able to perceive that there is still an inclination. This apparent change in the direction of a line viewed through a concave cylinder held obliquely, as it is moved from arm's length to the eye, could not be explained if Helmholtz' law of direction were true. If the axial ray were the line of direction, the apparent obliquity of a horizontal line would be the same whether the cylinder causing the phenomenon were held at arm's length or in contact with the eye, for the reason that, after the axial rays are deflected, some above and some below the horizontal plane, they pursue a straight course to the retina whether it be far away or near by. These rays prolonged, according to Helmholtz, would locate the source of the light, and necessarily would give it the same apparent inclination for all distances at which the cylindrical surface might be held from the eye. Not so with that law of direction which says that all lines of direction are radii of retinal curvature prolonged. This law makes it necessary for the line to appear to incline more when the cylinder is held far away from, and less when it is brought close to, the eye.

NOTES OF A CASE OF GLIOMA OF THE RETINA.

By Alfred J. Horsey, M. D., M. N. C. S., Eng.
M. R. C. S., Edin.

of Ottawa, Ont., Canada.

Surgeon to the Eye and Ear Department of the County of Carleton General Hospital, Ottawa.

Jane A., 4 years of age, the fifth child of a healthy family of six children. Father and mother healthy; no history of miscarriages. Two years ago she would occasionally awaken in great fright screaming and apparently would not know or be pacified by her parents. Had convulsions. With this exception her health was good. Six months ago a grayish yellow appearance was first noticed behind the pupil in the left eye.

On April 7, 1894. she was kindly referred to me by Dr. McKay, of Manotic, when the appearances in the eye were as follows: That which first attracted attention was a yellowish gray mass shining behind the dilated pupil, which could be easily seen with the unaided eye. It had a consistent look, and smooth, uneven surface, upon which there were no vessels. It half filled the vitreous chamber and appeared to proceed from the nasal side. The anterior chamber was shallow from pressure from behind; the iris was of the same color as the other; the pupil was dilated, but regular, reflex absent. The ciliary border was darkly congested by enlarged blood vessels, chiefly veins. Other blood vessels at the inner and outer canthus more superficial ran horizontally, the cornea and lens were clear. Tension + 2. Vision *nil*. Lids slightly edematous; no proptosis; no increase of corneal curvature; movement of globe normal; right eye normal. Immediate eneucleation was advised.

She was again brought to me on April 16, nine days after the first visit. The ciliary border was more prominent and more vascular, while the corneal curvature was increased and cornea steamy, with loss of luster. Tension about the same as before: + 2. Had occasionally cried, complaining of her head, the past two days.

April 18. Enucleation under chloroform; globe filled orbit so that it was with difficulty prized out intact. Nerve divided well back, looks healthy. A dark granular suspicious looking substance was removed from the upper part of orbit. Patient made a good recovery from the operation and remained in fair health up to July, three months after operation, when she began to fail. The orbit became full and the lids distended by a growth behind them, which gradually became more and more projecting until it protruded $2^1/_4$ inches in a cylindrical form with a tuberous summit, granular, and oozing an icherous pus and blood (fungus hematodes). The skin about the orbit was made to bulge forwards, the veins in which were greatly enlarged. She slowly declined during the remaining months of the year, being convulsed several times, until December 24, when she died—fourteen months after the discovery of the disease and eight months after operation. The right eye remained unaffected.

REMARKS.

I have thought the foregoing case sufficiently important for publication, not only as glioma is an uncommon disease and the only neoplasm which occurs in the retina, but on account of symptoms which would tend to confound it with pseudoglioma, which would materially modify the prognosis. Glioma is a growth from the neuroglia, and has its origin only in nerves or the nerve centers. It consists microscopically of small round cells, sometimes with spindle cells within a delicate stroma, securing very large neuclei. It is a disease almost exclusively of children, sometimes noticed at birth and frequently a few weeks or months afterwards. Though a rare disease, Messrs. Lawford & Collins (*Royal Lond. Hosp. Ophth. Reports*, 1890) publish sixty cases, fifty-five of which were cases in Royal Hospital during the years from 1871 to 1890, in connection with which many instructive points have been brought out. According to these observers, during the first two years of life is the period in which it oftenest shows itself. The disease may be bilateral, affecting both eyes simultaneously or within a short interval. The right and left eyes are about equally affected as also are the sexes.

Recoveries after enucleation are rare, even where the limit of three years is regarded as being such, the percentage being as low as 12% to 15%. Recurrence is the rule in a great majority, which terminate fatally in a few months. From the literature of the subject recoveries seem to bear little relation to the age of the patient, nor, strange to think, by the length of time between detection and removal. For example, in a case of undoubted glioma there was no return after nineteen years, disease was detected at 2 years of age and was not operated on till one and one-half years later.

Diagnosis in advanced cases may be made with tolerable certainty, but in their beginning, if then detected, diagnosis is uncertain and difficult. The microscope alone can decide its real character. That often while it is most difficult to differentiate, it is pseudoglioma, which does not mean any particular and definite pathological condition, but any resembling glioma which might be mistaken for it. The difficulty of diagnosis may be more readily estimated when it is known that of twenty-four eyes removed for supposed glioma retinæ at the Royal London Ophthalmic Hospital (Moorfields), whereon a consensus of opinion of wide experience was brought to bear, notwithstanding which, seven subsequently by microscopic examination showed a mistaken diagnosis. Other conditions likely to be confounded with glioma are persistent fetal conditions, notably persistency and potency of the hyaloid artery, causing excessive and maldevelopment of the lens capsule. Also concretions of tubercle in the choroid. Detachments of the retina from other growths, or the products of ophthalmitis.

In the above reported case the cerebral symptoms preceding the disease in the eye, viz., the startling cry out of sleep and convulsions, which were indicative of meningitis, rather pointed to pseudoglioma than true glioma, the latter of which a microscopic examination proved it to be.

There was not at any time middle-ear disease or a purulent discharge from the ears.

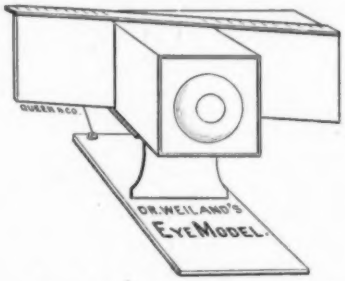
A NEW ARTIFICIAL EYE, MADE ENTIRELY OF GLASS AND GIVING IMAGES OF EXACTLY THE SAME SIZE AS THE SCHEMATIC EYE OF HELMHOLTZ.

By Carl Weiland, M. D.

CHIEF CLINICAL ASSISTANT, EYE DEPATMENT, JEFFERSON MEDICAL COLLEGE HOSPITAL, PHILADELPHIA.

IT is the purpose of the following lines to describe an artificial eye, the refractive part of which consists entirely of glass, and which is so constructed that the images formed by it are of exactly the same size as they are in the emmetropic human eye, or more correctly expressed, as they are in the latest schematic eye of Helmholtz. As the size of the retinal image depends upon the distance of the second nodal point of the eye from the retina, which in the latest eye of Helmholtz equals 15.49 *mm.*, the new eye must be so constructed that its second nodal point is likewise 15.49 *mm.* from the retina. As, furthermore, only *one* refractive surface is necessary in imitation of the reduced eye of Listing and Donders, and as one refractive medium allows the nearest approach to the human eye, we have taken only *one* refractive medium, glass, which enables us to find the radius of curvature that has to be given to the refractive surface in order to comply with our demands. For it is known from the theory of refraction that in one refractive medium the two nodal points are reduced to one and coincide with the center of curvature of the anterior surface of the refractive medium. It is further known that the distance of this nodal point from the second principal plane or retina is equal to the anterior focal distance F_1 of this reduced eye, which gives the equation $F_1 = \frac{r}{n-1}$, where r is the radius of curvature of the refractive surface and n is the refractive index of glass (in our case = 1.53). As F_1 is to be equal to 15.49 *mm.*, we have $15.49 = \frac{r}{1.53-1}$, which makes $r = 8.20$. All we have to do, therefore, is to grind a small plano-convex lens of a radius of 8.20 *mm.*

and to cement this with Canada balsam (of the same refractive index as glass) to a block of the same glass as the accompanying illustration shows. The length of the block of glass must be such that its posterior surface coincides with the posterior focal plane of the eye, which means that the distance from anterior surface of cornea to retina must be $r + \frac{r}{n-1} = 8.20 + 15.49 = 23.69$ *mm.* The posterior focal distance of this new reduced eye is therefore 23.69 *mm.* But as it is almost impossible for the manufacturer to exactly obtain this distance of 23.69 *mm.*, and as it is further desirable that the eye should be made longer or shorter to represent the axial changes of the human eye, a prism of glass (of 10° angular aperture) is brought in contact with the posterior surface of the eye, the plane of which has been ground off obliquely, so



that it makes an angle of 80° with the optical axis of the eye. For the addition of the prism again produces a surface, which is always at right angles to the optical axis; while by pushing the prism along the posterior surface of the glass block the eye can be made longer or shorter without the introduction of air.

A scale has been added, which exactly indicates the refractive condition of the artificial eye. This scale is calculated with this supposition, that the refraction is reckoned in the same way as in our own eye, namely, from the anterior focal point of this eye, which lies 15.49 *mm.* in front of its cornea. Now the scale can be easily calculated just as in the human eye; for in such a system of an eye with axial emmetropia and correcting lens at its anterior focal point the absolute values of the anterior and posterior focal distance remain the same as in the emmetropic eye, and so, also, does the distance of second nodal point from retina or second principal focus, but this nodal point is displaced to the same extent as the second principal plane, namely, by $-\frac{F_1 F_2}{f}$ *mm.*, where F_1 and F_2 refer to the anterior and posterior focal distances of the eye, and f indicates the focal length of the lens, used at the

anterior focal point of the eye to correct the axial ametropia. In our new eye we have $F_1 = 15.49$ *mm.* and $F_2 = 23.69$ *mm.*, and if we now take f as equal to 1000 *mm.*, *i. e.*, if we take a lens of 1 dioptry, we find that the axial lengthening of our new eye, to be corrected by a glass of 1 D., must be $-\frac{15.49 \times 23.69}{1000}$ *mm.* $= -0.367$ *mm.* If, therefore, we displace the retina of this eye in the direction of the optical axis by 0.367 *mm.* we obtain a difference of refraction equal to 1 dioptry. This is almost exactly the same as in the human eye, or rather in the average eye of Helmholtz, where a displacement of the retina equal to 0.321 *mm.* gives a difference in refraction equal to 1 D. But as in the new eye the prism is moved obliquely it takes more than 0.367 *mm.* in the direction of its movement to get 0.367 *mm.* in the direction of the optical axis. A little calculation shows that the prism must be moved by $\frac{0.367}{\sin 10^\circ} = 2.1$ *mm.* for each dioptry. The posterior surface of the prism consists of ground glass, so that we may observe the retinal images of objects which are now of the same size as in the human eye.

By making the eye ametropic we may observe the images of ametropic eyes, which will appear in diffusion circles of about the same size as in the human eye. The diameter, *d*, of the diffusion circle in the new eye for axial ametropia equals: $d = p \frac{F1 \; F2 \; D}{1000 \; (F2 - m)}$ where p is the diameter of the artificial pupil, F_1 and F_2 the anterior and posterior focal distances of the new eye, m the distance of pupil from cornea and D. the refractive value of the glass which would correct the ametropia. The diameter (d_1) of the diffusion circles of the schematic human eye according to Nagel[1] is $d_1 = \frac{p \; F1 \; D}{1000}$, where F_1 is the anterior focal distance of the human eye and p and D have the meaning before given. Now as the ratio of $\frac{d}{d1} = \frac{F2}{F2 - m}$ as F_1 of our artificial eye $= F_1$ of the human eye, by construction, we see that the diffusion circles are slightly larger in the new model, but not very much. For example an axial myopia of 1 dioptry gives in the schematic eye a diffusion circle of 0.06 *mm.*, while that of the new eye is 0.07 *mm.*

The ametropia may be corrected by a lens in front of the stop, which indicates the anterior focal plane of the model. There is besides a spring at the back, which allows any picture of the fundus to be brought in contact with the prism, where it can be viewed with the ophthalmoscope at the stop. In this manner the eye may be employed for *ophthalmoscopy*, *skiascopy* and *eidoscopy*, if I may be allowed to coin a new term for the act of observing

[1] *Die Anomalien der Refraction*, Graefe and Saemisch, *Capitel* X., p. 457.

the images produced by this eye. The scale will be very useful to check the observer. This eye may also be employed to obtain a correct idea about the size of a scotoma in a patient's eye; for all that is necessary is to bring the new eye at the place of the patient's eye, when the image of the boundary of the scotoma, which must have been marked off in a black wall before, will appear of almost the same size as it is in the patient.

Let us now compare the new eye with that of Helmholtz:

The Schematic eye of Helmholtz has:	New Reduced Eye of Glass has:	Difference.
Cornea = 7.8 *mm.*	Cornea = 8.2 *mm.*	+ 0.4 *mm.*
Anterior focal distance = 15.5 *mm.*	Anterior focal distance = 15.5 *mm.*	0 0 *mm.*
Distance of anterior focal point from *cornea* = 13.7 *mm.*	Distance of anterior focal point from *cornea* = 15.5 *mm.*	+ 1.8 *mm.*
Distance of posterior focal point from cornea = 22.8 *mm.*	Distance of posterior focal point from cornea = 23.7 *mm.*	+ 0.9 *mm.*
Distance of second nodal point from retina = 15.5 *mm.*	Distance of nodal point from retina = 15.5 *mm.*	0.0 *mm.*
Axial lengthening or shortening required to produce an ametropia of 1 dioptry as measured by a glass at anterior focal point of eye = 0.321 *mm.*	Axial lengthening or shortening required for the new eye under the same conditions = 0.367 *mm.*	+ 0.046 *mm.*
Length of eyeball from anterior surface of cornea to posterior surface of sclera = 22.8 + 0.9 *mm.* = 23.7 *mm.*	Length of eyeball from anterior to posterior surface = 23.7 *mm.*	0.0 *mm.*

From the table it will be observed that this new eye in more than one respect differs very little from the schematic eye of Helmholtz, but that as far as the size of the retinal image is concerned it is exactly like it. In short it is a *reduced eye* in glass. The advantages which this eye offers over other artificial eyes are the following:

1. It gives images of the same size as the average human eye.
2. It may be used for ophthalmoscopy, skiascopy or eidoscopy.
3. It gives a good idea about the small change in the length of an eyeball that is necessary to produce axial ametropia.*

* This artificial glass eye may be obtained from Messrs. Queen & Co., of Philadelphia, who have taken great pains in making it as accurate as possible.

SARCOMA OF CHOROID.

By Alfred J. Horsey, M. D., M. N. C. S., Eng.,
L. R. C. P., Edin.
of Ottawa, Canada.

Surgeon to the Eye and Ear Department of the County of Carleton General Hospital, Ottawa.

H. W., a well-grown, healthy looking lad, 16 years of age, having a good family history and no noteworthy personal history, consulted Dr. James Grant, in February last, on account of a smooth pultaceous tumor about the size and shape of the lateral half of a hen's egg, situated on the radial border of his right forearm, just above the wrist joint, which was opened and found to contain pus; there was also swelling and redness of the index, middle and ring fingers of his right hand at the roots of the nails, causing them to be clubbed, and which afterwards suppurated. In the front part of his left eye something abnormal was seen, which had been discoved by his friends some three weeks before, and on which account Dr. Grant kindly referred him to me.

On February 13, when I first saw him, a yellowish, well-defined body, 3 *mm.* square, having a smooth surface, was clearly seen in the front part of the left eye, which, at first sight, resembled an abscess in the cornea, but on closer examination with a lens was found to be a new growth in the anterior chamber, situated in the angle between the cornea and iris, extending horizontally inwards from the root of the iris on the temporal side, halfway across it towards the pupil, in a slightly radiating manner. Minute blood vessels running horizontally could be seen in its semi-translucent substance. The periphery of the iris was slightly dragged upon giving the impression that the growth came from behind it. The

pupil was slightly larger than its fellow and sluggish to light; the ciliary zone was congested. R. V. = $\frac{6}{9}$; L. V. = $\frac{6}{5\,6}$. Tension slightly increased. Ophthalmoscope — Fundus reflex indistinct by direct method only; large vessels were obscurely seen in the fundus, for a short distance, then lost in darkness.

February 15. Two days later vision had so far diminished that he could barely read $\frac{A}{6\,0}$. Tension as before. A disc of homatropin and cocain only slightly dilated the pupil. His eye caused him no pain, and only slight inconvenience, though vision was so greatly impaired.

The diagnosis, "sarcoma," found at his first visit, was further strengthened at his second, and immediate excision of the eye advised. He was recommended to get another opinion before the operation, which he did, and which coincided with my own. The eye, however, was not excised until a month later, when a microscopic examination confirmed the diagnosis of sarcoma. The patient made a fair recovery from the operation, but after a couple of weeks rapidly lost flesh, became cachectic, complained of dizziness, and a few days before his death, which occurred on May 5, he had several attacks of right-sided spastic convulsions. No post-mortem was obtained. Probably the cause of death was extension backwards by continuity from the choroid to the pia mater and involvement of other organs by metastasis. The temperature was not much disturbed and showed no great fluctuation. The other (right) eye was not affected.

The case is instructive in several particulars. Sarcoma of the choroid being a disease of retrograding adult life, occurring most frequently beyond the age of 50, the youthfulness of the patient makes it a rare exception.

Sarcoma of the choroid being a disease of adult life, contrasts strongly with glioma retinæ, another intra-ocular malignant growth, occurs almost exclusively in early childhood. That it was without pigment, which is exceedingly uncommon in growths in this highly pigmentus covering. That it was so rapidly fatal — its duration being measured by months rather than years — two years being the average length of life in fatal cases. Prognosis is not nearly so unfavorable as in glioma retinæ, where recovery is rare. In sarcoma the mortality, according to several high authorities, is about 50%, 32% being from metastasis to distant organs. That it was idiopathic; no injury or previous disease in the eye, as is often the case. That it was unattended by pain.

A CASE ILLUSTRATING THE RELATION BETWEEN THE MUSCULAR BALANCE OF THE EYES AND THEIR REFRACTIVE CONDITION.

By C. E. Norton, M. D.,
LEWISTON, MAINE.

OPHTHALMIC SURGEON TO THE CENTRAL MAINE GENERAL HOSPITAL.

MISS E. P., a student in Normal School, 20 years of age, consulted me August 23, 1893, in regard to her eyes. She was in good general health. She had a noticeable convergent strabismus in the right eye. Vision in right eye = $\frac{20}{40}$, in the left eye = $\frac{20}{30}$.

She had been wearing for several months a pair of — 1.25 D. spherical spectacles which she had obtained from a prescribing optician. The use of these spectacles caused the strabismus to disappear, but it returned almost immediately on removing them. She did not wish to have an operation performed, but she wished me to prescribe for asthenopia from which she suffered severely when studying.

I found that she had hyperopic astigmatism which could be corrected by + 0.25 D. cylinder axis 90°, both eyes. I prescribed these lenses for constant use. With them her vision was $\frac{20}{20}$ in each eye. With the aid of these spectacles she was able to finish her course of study, and she graduated from the school in the spring of 1894. Since graduation she has been teaching in a city school. She called at my office March 23, 1895, and stated that if she wears the glasses constantly she has no return of the strabismus, and is able to do her work without discomfort in her eyes. Going without the glasses causes the strabismus to return after a time.

The above case has several points of interest. It will be noticed that the vision was good in both eyes, and that in the squinting eye it was but very little less than in the other eye. There was no marked amblyopia to account for the strabismus, and we have every reason to believe that in this case the sole cause of the strabismus was the optical error, acting on an overworked eye; the natural relation between convergence and accommodation producing the result. When this relation was disturbed, by the use of concave glasses, increased convergence ceased to give the same assistance to the power of accommodation, which it did before, and the convergence disappeared.

The case is interesting on account of its rarity. Cases are sometimes seen where strabismus exists, accompanied with, and probably caused by optical error in which the strabismus entirely disappears, from no other treatment than the use of properly fitting spectacles.

I have never seen a case before, nor do I remember of reading of one, where a strabismus caused by hyperopic astigmatism, was made to disappear by the use of concave spherical lenses.

The case is also of interest by being a positive contribution for the solution of the question which has been under discussion for the last few years: "Are weak lenses of any value?" It is evident that in this case weak lenses were of great value, as they relieved the asthenopia, and, what is of more importance, the strabismus, without any operation being performed.

THE QUARTER DIOPTRY CYLINDER—SOME TESTIMONY FOR.

By E. E. Hamilton, M. D.,
of Wichita, Kan.

THAT the complexity of symptoms known under the general term "asthenopia" has its cause, in a large majority of cases, in ciliary muscle strain, all agree. No fact in ophthalmology is more firmly established.

Errors of refraction, both axial and those due to irregularities of corneal or lenticular curvature, (unequal refraction in different meredians of the eye) impose additional work on the ciliary muscle. Under such conditions, in many instances, the overtaxed muscle gives way under its almost constant tension and causes distressing symptoms located in and around the eyes.

Perfect health may, and often does, compensate for these errors. Most frequently the patient's discomfort dates from an overuse of the eyes, or some impaired state of the health.

Often too the excessive muscular effort in accommodation disturbs the relation between it and convergence. Here a loss of muscular equilibrium may add another factor to the patient's unpleasant sensation, in extra ocular muscle strain. However much we may differ as to the part that aberrations of the extrinsic muscles have to do in the production of eye strain, it is a fact patent to us all that the nearer the eyes are brought to a condition simulating emmetropia, the less work is imposed on the muscular machinery concerned in the maintenance of binocular single vision.

While there is a unanimity of opinion that the grosser refractive errors, particularly astigmatism, cause these asthenopic symptoms, there still exists in the minds of a minority contingent of the profession at least, some skepticism as to any beneficial effects resulting from the correction of a low degree of astigmatism measured by the quarter dioptry cylinder.

Personally I am convinced these slight imperfections in corneal curvature often are fruitful sources of eye strain, including headaches. That I may contribute my mite in favor of the quarter dioptry cylinder, I wish to report fifty cases of asthenopia in which this glass has been the principal, in most cases, the only treatment. In a very few cases I have prescribed a collyrium for a conjunctival congestion or an ointment for a slight blepharitis, but even here the error of refraction has been considered the cause, and these local troubles manifestations of eye strain. In no case did any condition of the patient's health or extrinsic muscles seem to demand special attention. All were under 40 years of age and all were corrected with a suspended accommodation.

In collecting data for this report I have talked personally with eleven and addressed the following question to thirty-nine patients: "For what symptoms did you consult me, and what, if any, relief have you gotten from your glasses?"

Of the eleven with whom I talked ten reported satisfaction and one dissatisfaction with the glasses. From the thirty-nine letters I have received thirty-one answers, leaving eight of the whole fifty cases unheard from. Twenty-nine answered favorably, all expressing satisfaction of different degrees, with their glasses. Two answered negatively. Of the eight who failed to answer two letters were returned to the sender, their address not being the same as when they visited me. The remaining six failed to answer, either from indifference or perhaps were dissatisfied without wishing to so express themselves.

At the risk of being tedious, I wish to report a few typical cases in full. To report them all would tax your patience too much. I have made no attempt to arrange cases in groups according to symptoms, the answers being so varied it seemed difficult so to do.

Case I. Mr. B., farmer, 36 years of age, was given May 25, 1893, glasses as follows: R. E. cyl. + 0.25 D. ax. 60°; L. E. cyl. + 0.25 D. ax. 115°, with directions to wear them constantly during waking hours. While doing some work for a member of Mr. B's family, he asked if I would recommend him to a good physician, as he wished to consult him about his headaches. I suggested his eyes as a possible cause and asked him to allow me to examine them. The above glasses was the result of my examination. December 6, 1894, he says: "Since my earliest remembrance I have averaged two days a week in bed with sick headache. Never suspected my eyes as a cause. I saw well and they

gave me no pain. Since putting on my glasses, eighteen months ago, I have had headaches, but not oftener than once a month at any time. Haven't had any sickness of my stomach. My headaches are getting less and less frequent, and very much lighter. I wouldn't trade my glasses for the best farm in Kansas." February 23, 1895, he called to inform me he had been without his glasses ten days, having broken his frames. Four days after he ceased wearing them he had one of the worst attacks of sick headache he ever had. Was in bed forty-eight hours. The headache and nausea were terrible.

Case II. Prof. H., 35 years of age, principal of public schools of Newton, Kansas, consulted me June 23, 1894. He was given R. E. cyl. + 0.25 D. ax. 45°; L. E. cyl. + 0.25 D. ax. 135°, with instructions to wear the glasses constantly. January 3, 1895, he writes: "I was troubled so much with nervous headache I couldn't read ten minutes without a severe pain in my eyes and head. While I despise wearing glasses, I must say I couldn't get along without them. They have helped me so much. I can now read for hours at a time without feeling much pain or weariness from my reading."

Case III. Miss H., 18 years of age, consulted me August 3, 1894. Under ophthalmic discs of homatropin and cocain, she would not accept any spherical glass. After repeated trials I prescribed R. E. cyl. — 0.25 D. ax. 90°; L. E. cyl. + 0.25 D. ax. 90°. She had worn B. E. sph. + 0.50 D. given her by another oculist. January 3, 1895, she writes: "I could use my eyes but a few minutes until they would ache so badly I had to stop my work. Could not pursue my school studies. The glasses have helped me very much indeed. I have attended school since September and have had no trouble to keep up with my classes."

Case IV. Mr. S., 27 years of age, consulted me March 18, 1893. I prescribed R. E. cyl. + 0.25 D. ax. 180°; L. E. cyl. — 0.25 D. ax. 180°. December 6, 1894, he reports as follows: "My principal complaint was 'car sickness.' I couldn't go to Newton without having a terrible headache. My business frequently calls me to New England and I dreaded the trips. While riding at night I would have no trouble, but in the day time, if I kept my eyes open, I would suffer terribly. I have just returned from such a trip and I wore my glasses every waking minute, and I haven't had a headache during my whole trip."

Case V. Edna D., 10 years of age, was referred to me by Dr. J. E. Oldham for an examination of the eyes to see if I could find

cause for obstinate headaches. Finding astigmatism with greatest corneal curvature horizontally, I prescribed (August 6, 1894,) B. E. cyl. + 0.25 D. ax. 180°, and told her to wear her glasses all the time. January 5, 1895, her mother reports: "Headaches very much improved. Has hardly complained at all for the last month. Goes to school steadily."

Case VI. Mrs. S., 26 years of age, consulted me about her eyes May 1, 1893. She complained of some discomfort on use, but principally of a succession of styes that were most annoying. I gave her B. E. cyl. + 0.25 D. ax. 90°. January 10, 1895, she wrote: "I never read or sew without my glasses. I have had but one stye since. They have benefitted my eyes greatly."

Case VII. Mrs. P., 23 years of age, was given B. E. cyl. + 0.25 D. ax. 90°, June 9, 1894. January 9, 1895, she wrote: "My principal complaints were painful, burning sensations in eyeballs. I couldn't use my eyes any length of time without causing me to have severe headaches. They were very sensitive to light, often had a dimness of vision while using them; could scarcely see at times. I have had no headache since I got my glasses. I am compelled to wear them all the time, as it seems as if the light was too strong without them. I have gotten much relief from my glasses for which I am very thankful."

Case VIII. Inez N., 12 years of age, was given B. E. cyl. + 0.25 D. ax. 180°, March 26, 1894. January 2, 1895, Mrs. N. writes me: "Inez complained of blurring of words when reading, and of objects when looking steadily at them, and of darting pains in the eyes. Since she commenced wearing her glasses she never has complained of pain or blurring."

Case IX. Miss P., 22 years of age, was given B. E. cyl. + 0.25 D. ax. 180°, August 10, 1894. January 2, 1895, she states: "I have been greatly benefitted by my glasses. The headaches I suffered so much from have been very greatly relieved. My eyes do not ache now. I am very much pleased with the glasses."

Case X. Miss S., 19 years of age, was sent to me by Dr. Sippey, of Garden Plain, Kansas. She was given B. E. cyl. + 0.25 D. ax. 180°, May 13, 1893. January 7, 1895, she writes: "My trouble was headache. I wore the glasses for nearly a year, and am sorry to say received no benefit from them."

Case XI. Miss W., 22 years of age, was fitted with glasses June 9, 1894, R. E. cyl. + 0.25 D. ax. 105°; L. E. cyl. + 0.25 D. ax. 75°. January 19, 1895, she writes: "Have worn the glasses constantly. They not only have relieved my eyes of that

aching feeling, but have rendered them so strong that I use them in my work as a teacher, without fatigue."

Case XII. Mrs. H., 25 years of age, was sent to me by Dr. Spitler February 10, 1894. She was given R. E. cyl. + 0.25 D. ax. 60°; L. E. cyl. + 0.25 D. ax. 120°. January 8, 1895, she writes: "My eyes would pain me very much and become red and inflamed on use. When I first commenced wearing my glasses objects seemed tilted one side and the floor did not seem level. After one or two weeks these sensations passed away. While my eyes still trouble me some, my glasses are of great assistance in reading or sewing."

Case XIII. Mr. H., 38 years of age, farmer, referred to me by Dr. Burton for relief from aching eyes and head. November 14, 1893, he was given R. E. cyl. + 0.25 D. ax. 135°; L. E. cyl. + 0.25 D. ax. 45°. January 14, 1895, he reports: "I suffered from sick headache once or twice a month for years. For past year could use my eyes but a few minutes at a time; they would itch and burn, and the light, both natural and artificial, would give me great distress. Had to shade them almost constantly. Have worn my glasses two months. For a few days when I first began wearing them I felt as though I was standing on my head, they made me so dizzy. I persisted in wearing them constantly, and now they are all right. I haven't had a headache since, and can read by lamp light two or three hours without discomfort."

Case XIV. Mr. J., 38 years of age, consulted me October 6, 1894, about a severe blepharitis marginalis and conjunctival congestion, stating that he had spent much time and money trying to get a cure, but he had failed. Had never had glasses suggested. He declared his eyes never gave him any discomfort, though the redness of his lid margins was so conspicuous as to amount almost to a deformity. I ordered B. E. cyl. + 0.25 D. ax. 180°, advising him to wear them all the time; also gave him an eye wash and yellow oxid of mercury ointment. January 15, 1895, he writes: "My eyes are slowly improving. Think if I would wear my glasses all the time, as you advised me, they would get well. I only wear them in close work, disliking so much to wear them at other times. They certainly have greatly benefitted my eyes."

Case XV. Miss F., seamstress, 19 years of age, referred to me November 29, 1893, by Dr. Hupp for an examination of eyes for headache, which he had been unable to relieve. I prescribed B. E. cyl. + 0.25 D. ax. 90°. January 7, 1895, she informs me:

"My eyes are easy and I have no more headaches so long as I wear my glasses, but when I try to do without them there is trouble."

Case XVI. Mr. M., 28 years of age, student, was given September 1, 1894, B. E. cyl. + 0.25 D. ax. 180°, hoping to relieve obstinate asthenopic symptoms, and a conjunctival congestion. January 5, 1895, he writes to me: "The result of your work is not entirely satisfactory. Since I commenced using the glasses I find it quite impossible to do without them. At present I am able to do three hours' more work a day than I could before I received the glasses. The redness of my eyes sometimes disappears entirely; at other times they get very red."

Case XVII. Miss A., dressmaker, 23 years of age, sent me by Dr. Fabrique, was given, February 3, 1894, R. E. cyl. + 0.25 D. ax. 90°; L. E. cyl. + 0.25 D. ax. 135°. January 2, 1895, she reports: "I cannot do without my glasses, even for distance. Without them my eyes tire and blur, and get red. I wouldn't think of doing without them."

Case XVIII. Miss D., 21 years of age, consulted me December 18, 1893, for headache and uncomfortable eyes, caused by sewing or reading. I ordered R. E. cyl. + 0.25 D. ax. 135°; L. E. cyl. + 0.25 D. ax. 45°. January 4, 1895, at my request, she called to inform me that she had not worn her glasses. She had tried, but they had made her dizzy, and she had given up trying to wear them. I examined her eyes again and found she accepted the same glasses. After encouraging her to give them a further trial she departed promising to report some future time."

Case XIX. Miss S., 19 years of age, school girl, referred to me by Dr. Fabrique. She was given, January 3, 1893, R. E. cyl. + 0.25 D. ax. 180°; L. E. cyl. + 0.25 D. ax. 165°. January 15, 1895, she writes: "I suffered continually from headaches and pain in my eyes, especially after reading or study. The glasses have relieved me entirely, and I anticipate no further trouble."

Case XX. Chas. C., 15 years of age, consulted me October 13, 1894, for "aching eyes," would water much on use. Examination showed about 5 D. of hypermetropia, and much amblyopia of right eye. Left eye accepted sph. + 0.50 D. ⊃ cyl. + 0.25 D. ax. 150°, giving him vision $\frac{20}{20}$. He was given a plane glass for right, and the above cylinder for left eye. January 15, 1895, he writes: "I go to school regularly, and wear my glasses all the time. While wearing them my eyes neither water or ache."

Case XXI. Walter H., 15 years of age, consulted me September 2, 1894, complaining much of headache, tired and watery eyes after looking at a book for a short time. He was given R. E. cyl. + 0.25 D. ax. 120°; L. E. cyl. + 0.25 D. ax. 90°. January 3, 1895, his father writes: "His difficulties seem to be entirely overcome by the use of his glasses."

I have no confidence in my ability to diagnose these slight degrees of astigmatism with any objective test. My reliance is a mydriatic, with trial case, test types and astigmatic card.

If there is any axial ametropia, I correct it with the spherical glass giving best vision, and then proceed with my cylinders; directing my patient's attention to the astigmatic card, resembling a wheel, I ask him if he discovers that each spoke contains three black lines and two white spaces? If he answers in the affirmative, as he no doubt will, I ask him to look carefully and see if he discerns the white spaces in each spoke equally distinct in all directions? If astigmatism is present, even of low degree, and the patient is a close observer, he will tell that in one direction the white spaces are indistinct or absent, giving an appearance of a solid black line. Adding now the correcting cylindrical glass at a proper angle, the spokes will all appear alike. When tested with Snellen's type at twenty feet, the same glass ought, and will, give the sharpest definition of vision.

Dr. Price, of Nashville, Tenn., has suggested that the exact axis of our glass can best be gotten with a stronger cylinder, a suggestion of value, which I have often put in practice.

In confirming results, I prize the crossed cylinder of Jackson, finding it of great assistance to the patient in determining the exact strength of the cylinder.

Dr. Prince, of Springfield, Ill., suggests the abandonment of the heavy trial frame with rotating cells which accompanies our trial cases. They have always seemed cumbersome to me, for which reason I do not use them. The simple cell is much lighter and more convenient. With it, by reversing their surfaces, cylinders with axes mounted at forty-five degrees, can be made to assume any angle.

SUMMARY: I found normal vision, without glasses, in forty-eight cases. Two cases had $\frac{20}{30}$, raised to $\frac{20}{20}$ with the cylinders. In both cases their apparent error was corrected with a minus cylinder, while the mydriatic correction required plus cylinders, of same strength, with axes at right angles. I always prefer the plus cylinder.

In two cases there were no symptoms whatever referable to the eyes; one of migraine, and one with blepharitis and conjunctival congestion.

Headaches were found a frequent symptom. With Dr. Chisholm, of Baltimore, who has so ably championed the quarter dioptry cylinder, I confidently expect to find astigmatism, oftenest of slight degree, in these headache cases, particularly if there is any associated complaint of eye distress. If headaches improve under a mydriatic, I confidently prognosticate relief from glasses. Here, as in all other cases, I much prefer atropin, having most confidence in its cyclophlegic properties, and liking too, the therapeutic effect of the prolonged rest given the eyes by that mydriatic.

There were numerous complaints of photophobia. It is a distressing symptom. Patients complain that both direct and reflected rays of light are painful, the former from their intensity; the latter probably from the acquired additional heat given them by our stone walks and asphalt pavements.

A frequent complaint from cylinders, with axes placed obliquely, comes from disturbed ocular perceptions, with consequent dizziness and sometimes nausea. In my experience, these sensations soon disappear under constant use of the glasses. On account of dizzy sensations, one of my patients ceased wearing her glasses before she had given them a fair trial.

Of my fifty cases, thirty-nine have reported satisfactory results, and three unsatisfactory. Counting the eight not heard from as failures, we still have left a percentage of success of seventy-eight.

In my humble judgement, the quarter dioptry cylinder has come to stay. So long as my patients continue to express relief from distressing symptoms, I shall continue to prescribe it. It is not "a superfluous placebo." On the contrary, in intelligently selected cases it is a remedy of great value both to the oculist and his patient.

127 N. Market Street.

TETANUS FOLLOWING WOUND OF LOWER EYELID.

By George F. Keiper, A. M., M. D.,

of Lafayette, Ind.

Ophthalmic and Aural Surgeon to St. Elizabeth Hospital, St. Joseph Orphan Asylum, etc.

EMMET N., 15 years of age, was brought to the office on May 18, because of double vision and inability to close the lids of the right eye. The right eyebrow was markedly arched. The orbicularis was paralyzed. The paralysis of the inferior oblique required a prism of 8° to neutralize it. On the lower lid, in its center, two lines from its edge, was a vertical scar about one half inch long, which he received from a wound made by a horse weed thrown by a companion while bathing in the river just nine days before. He said it required some force to pull it out, and that his companions picked out several pieces afterward. No attention was paid to it, however. Nothing further could be learned from his history. The next day his mother remarked that she had noticed him forgetful about certain matters to which no attention had hitherto been paid. Suspicion was immediately aroused that he had had an accident whereby "his head was hurt." That he denied, and his mother knew nothing of it. Since his death, a patient, a boy, told me he saw him fall very heavily from his bicycle four weeks before his death and strike his head.

The examination of the retinæ revealed no abnormality. The boy was weak and poorly nourished. I ordered syr. ferri iodid in 20 minim doses every four hours, and dilated his pupils with atropin to prevent him reading. The right eye was bandaged to avoid confusion. At 10:30 the next morning I was hastily summoned to his home, and I found him suffering severe pain, and I

was informed that he had slept none the night before. It gave him much pain to expose his tongue. I prescribed a mixture containing 10 grains each of bromid of potassium and chloral, to be given every hour until he slept, when it was to be discontinued. He took 5 grains of calomel also. At 5 o'clock, at the time of my visit, he was asleep. The bromid and chloral mixture was ordered continued as occasion required. The next morning, at 6 o'clock, his father hastily summoned me and arriving at the house, I found him in a condition of tetanus. The jaws were well set and the sternomastoids prominent, and both eyebrows markedly arched. The bromid and chloral mixture was directed to be given every half hour, and I requested that the family physician be called. Dr. Hupe and I met at 8 A. M., and the case was placed in his charge. The family requested me to continue attention to his eyes, which I did with a feeling of hopelessness. All the remedies usually used in such cases, especially the bromid and chloral, were pushed heavily. A consultation was held the same evening, with no hope expressed. The case continued thus until May 22, when he died at 5 A. M., during all of the time he was in a condition of opisthotonos frequently. The report of a similar case by Drs. Fromaget and Cabannes, of Bordeaux, is to be found in the *Annales d' Oculistique*, January, 1895.

GALLICIN, A GALLIC ACID DERIVATIVE—ITS USE IN THE TREATMENT OF EYE DISEASES.

By George F. Suker, M. D.,
of Toledo, Ohio.

THIS preparation of gallic acid, gallicin, was first used in ophthalmic practice by Dr. Carl Mellinger, Privat-Docent in the University of Basel, He has given it a fair trial, having used it for over a year, and finds it very efficacious in every respect. One main feature is its prompt action, then, too, it is void of untoward effects. That it might be indicated in certain catarrhal affections of the eye, is to be judged from its close relationship, chemically and therapeutically, to resorcin ($C_6 H_6 O_2$) and pyrogallol ($C_6 H_6 O_3$), which, as we know, are highly recommended for their efficiency in the treatment of catarrhal inflammations.

Gallicin is the methylic ether of gallic acid, and as such possesses the formula $C_6 H_2 (O H)_3 COO CH_3$. It is made by heating a methyl-alcoholic solution of gallic acid or tannic acid with hydrochloric acid gas or concentrated sulphuric acid.[1] When recrystallized from methylic alcohol it forms anhydrous rhombic prisms; and from hot water it crystallizes on cooling in the form of delicate, fleecy, snow-white needles. The latter being better adapted for therapeutic uses. Both preparations melt at a temperature of 200° to 202°. It is readily soluble in hot water, and in warm methylic and ethylic alcohols, and also in ether; the solution being colorless. Gallicin is more preferable than pyrogallol in that it possesses no poisonous properties. It is generally used in a powdered form, and is applied as we apply calomel, *i. e.*, with a fine camel's hair brush, or simply dusted on to the parts. Applications are usually made twice daily, though once will often suffice.

[1] *Correspondenzblatt für schweizer Aerzte*, Vol. XXV., p. 231.

Occasionally after the application of gallicin, the patient experiences a burning sensation, which can be partially allayed by a cold douche, or entirely relieved by the instillation of a few drops of a 2 per cent solution of hydrochlorat of cocain.

Dr. Mellinger has tried it in about 200 cases, and makes a most favorable report of its use. He says it is applicable in any form of catarrhal conjunctivitis, and especially is it of value in those cases where there is a chronic swollen condition of the mucous membrane with a more or less ropy discharge accompanied by an eczematous condition of the palpebral edge. In this class of cases its two-fold action comes into play, *e. g.*, for the catarrhal conditions, and for the skin affections.

Again, it is of great service in cases of catarrhal affections following septic infection, or in severe inflammation, such as is seen in the subsiding of a panophthalmitis or hypopyon keratitis. Furthermore, gallicin is of benefit in the follicular form of conjunctivitis, either acute or chronic. It is of value after cataract extraction, if a catarrhal condition supervenes, the troublesome secretion ceasing after four or five applications.

The close relationship it bears to resorcin and pyrogallol suggested its use in eczematous conditions of the eyelids for the relief of which it has proved very efficacious. In cases of phlyctenular conjunctivitis it is noticeable that after one or two applications of gallicin, the peripheral phlyctenules disappear rapidly. Gallicin is fully as good, if not better, than calomel in phlyctenular affections with copious secretions. In such cases calomel has a tendency to increase rather than decrease the discharge. In superficial keratitis, gallicin is of great value, as it renders a prompt resolution and clearing up of the cornea. I have confirmed these observations in the treatment of thirty cases. We can sum up the therapeutic value of gallicin as follows: It is applicable in all cases of catarrhal affections of the mucous membrane of the eye, either with or without secondary eczema; in cases of phlyctenular keratitis or conjunctivitis; and exceedingly serviceable in follicular conditions, and in superficial keratitis.

Gallicin is indeed worthy of trial, and in the future will, no doubt, play an important role in the treatment of the above named diseases. It is best employed in the powdered form; and, being very light, about 1 centigram is sufficient for an application. It is to be applied once or twice daily as deemed expedient.

REPORT OF A CASE IN COUNTER EVIDENCE TO THE INFECTIOUS THEORY OF SYMPATHETIC OPHTHALMIA.[1]

By H. L. Hilgartner, M. D.,
of Austin, Texas.

IN spite of the numerous observations and experiments on the subject, we are to-day far from clear as to the pathogenesis of sympathetic ophthalmia. There was a time when this problem seemed solved beyond a doubt by Deutschmann's investigations, but during the past few years his theory has met with so many objections in Germany, France, England and America that a doubt as to the value of his results can no longer be overcome.

Nearly fifty years ago William MacKenzie summed up his conception of sympathetic ophthalmia in the following words: (MacKenzie, Diseases of the Eye, Fourth Edition, p. 597, 1885). "I think the chief medium through which sympathetic ophthalmitis is excited is the union of the optic nerves." This theory found most general acceptance among ophthalmologists, until the appearance of Heinrich Müller's investigations fifteen years later. Müller[2] endeavored to find the channel of communication in the ciliary nerves. His conclusions were drawn from the anatomical examination of three eyes which had been enucleated through fear of sympathetic disease in the fellow eye. While conceding the possibility of inflammatory transmission along the optic nerves, "the irido choroiditis of the first eye," he says, has progressed so far that advanced atrophy of the optic nerve is present. The nerve is nothing more than a fibrous cord, incapable longer of conducting an irritation, or indeed any other process, so that simply cutting through the optic nerve will not lessen the chances of sympathetic trouble. The ciliary nerves, on the other hand, do not easily atrophy.

[1] Read before the Texas State Medical Association, April 25, 1895.

[2] *Graefe's Archives* Iv., p. 367-370.

The majority of eye diseases attack the anterior half of the eye, and consequently the ciliary nerves from their position would be more exposed to irritation. And when the inflammation of the second eye makes its appearance under the garb of irido-choroiditis as it frequently does, it is far more logical for us to assume that the inflammation was brought about through the ciliary nerves than through the optic nerve. It is not improbable that the ciliary nerves exercise some direct influence upon the nutrition of the retina and optic nerve.

Muller's ciliary nerve theory seems to me to find confirmation in the following case in my own experience:

In September, 1894, Wm. McD., 14 years of age, came to the Blind Institute at Austin, Texas, with the following history: Twelve years ago he stuck a knife blade into his left eye, causing loss of sight in the wounded organ within three days. From the time of the infliction of the injury until the patient came under my notice, he suffered intervals of pain from the injured eye, which were attended by sensitiveness in the fellow eye. Examination disclosed that the left ball was very much shrunken, besides being abnormally sensitive to light and touch. A cicatrix was found extending from the sclero-corneal junction to the center of the cornea. Upon first examination I noted patient lowered his head continually to avoid as far as possible exposure to light, complaining at the same time of pain in the unhurt eye. Upon close examination faint ciliary injection was discovered in the latter organ. The patient was advised to have the left eye removed, and on the following day the operation was performed. The stump being removed and examined, revealed a calcareous condition of the interior, including everything within the sclerotic coat. The right eye was promptly treated with atropin, and patient remained in dark room for several days, the treatment being continued until all apparent inflammation had subsided and recovery seemed secured. Two weeks later the patient attempted to read, with the result that on the day after, the pain in the eye returned, and upon examination ciliary injection was again disclosed. The pupil was very much contracted and did not respond to light. The treatment formerly pursued was repeated with the addition of hot fomentations, and after ten days recovery seemed complete, the atropin being continued, however, for one week thereafter.

During the second attack in the good eye, the empty socket was carefully examined and revealed no cause for the irritation in the fellow organ. Between four and five weeks after atropin had

been discontinued, the patient again attempted to read, with the same result, except that the new attack was far more tedious and stubborn than the preceding one, although it yielded to treatment after two weeks. The patient continued comfortable for one month after the ciliary muscle regained its power, when, though cautioned not to attempt as yet to use the eye, the injunction was disobeyed with a result far more serious than before, and for a time seemingly hopeless of remedial treatment. After the subsidence of inflammation, realizing the danger of any similar indiscretion on the part of the patient, the eye was kept continually under the influence of atropin for two months. After the lapse of several months since the recovery of ciliary action the eye now seems to be entirely normal.

Before the removal of the diseased eye, sympathetic irritation had begun in the ciliary nerves, as evidenced by the repeated inflammatory symptoms following ciliary action of the right eye. It seems therefore evident from the subsequent history of the case, that the enucleation of the diseased eye was not adequate to the removal of all irritation in the ciliary nerves, for that irritation manifested itself with continually increasing violence on repeated subsequent excitations.

It seems clear that the diseased condition of the nerves remained dormant for a while, and so far from manifesting a tendency towards abatement, only awaited the occasion for its development.

The counter theory of Deutschmann and his school would explain the case above cited on the hypothesis of parasitic infection.

The reasonableness of this theory of an infectious origin for sympathetic ophthalmia, no doubt accounts for its acceptance; yet this theory owes almost its entire support to a few experiments with pus organisms. Now it is well known that the pus organism does not produce sympathetic ophthalmia, as is proven in panophthalmitis when these organisms, though most abundant, do not as a rule give rise to sympathetic ophthalmia. Deutschmann's[3] views depend upon the bacteriological examination of seventeen eyes, which had caused sympathetic ophthalmia, and in every case, save one, organisms were found, and as regards this (one) negative result, he says: "It proves that no organisms were found in my sections and does not prove that there were no organisms anywhere present in the eyeball." In the majority of cases these microorganisms were found also in the optic nerve and its sheaths.

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[3] *Archiv. Ophthal.*, Vol. XXII., p. 303, 1893.

Wangemann demonstrates the presence of organisms in two eyes which had given rise to sympathetic ophthalmia.

Deutschmann's work has been repeated by Alt, Gifford, Mazza, Randolph, Ulrich, Limbourg, Levy and Richard Greef, but every one got only negative results.

Richard Greef[4] says: "A systematic examination was gone through in five cases where sympathetic ophthalmia had developed, and notwithstanding the most careful bacteriological examinations according to the various methods, no organisms were found." Similar results are reported by Nordenson, Ayres, Alt, Berry, Schmidt-Rimpler, Trousseau, Poncet and Uhltoff.

Deutschmann[5] rarely failed to find organisms in an eye enucleated to avoid sympathetic trouble. Randolph examined no less than a score of such eyes, and succeeded in detecting organisms in but one case and that was when the injury dated only two weeks back; a fact which seems to warrant the conclusion that the very questionable agency of micro-organisms in the production of sympathetic ophthalmia is rendered more unlikely when the sympathetic disturbance makes its appearance from two to six months after the injury.

Ohlmann[6] records thirty eyes enucleated to avoid sympathetic trouble, and in not a single one did he find organisms.

Cases in which a considerable period elapsed between the injury and the outbreak of the sympathetic inflammation can hardly be accounted for on this theory for the micro-organisms in the optic nerve of the injured eye would have perished long before the appearance of the disease in the injured eye.

Richard Greef[7] examined the ends of optic nerves in fourteen cases where neurectomy was made for fear of sympathetic disease, and in not a single instance did he find organisms, either in the optic nerve or its sheath.

Innumerable other investigations might be adduced, which, like the above, fail entirely to verify the conclusions of Deutschmann, and point rather to the positive conclusion that the micro-organism is in the majority of cases not present as an exciting cause.

Collecting the results of our investigation, it would seem clear from the case above cited that the lapse of twelve years between the injury and the outbreak of the sympathetic trouble in the other

[4] *Archiv. Ophthal.*, Vol. XXII, p. 302, 1893.
[5] *Archiv. Ophthal.*, Vol. XXI., p. 373.
[6] *Archiv. f. Augen.*, Bd. 22, 1.
[7] *Archiv. Ophthal.*, Vol. XXII., p. 303.

eye must have annihilated any micro-organic agency as a factor in the case. Such a period of time far transcends any scientifically established term of life for such organisms, nor does it seem possible that their operation would have remained for so long a time in abeyance, even on the assumption of their presence in the system.

In conclusion, it seems most probable that the pathological symptoms in the uninjured eye resulted from a purely mechanical irritation, propagated from the diseased organ before its removal, and asserting itself after that removal with steadily increasing violence until rest and treatment restored the normal state.

RELATIONSHIP BETWEEN DISEASES OF THE EYE AND BRAIN.[1]

By Robert Fields LeMond, A. M., M. D.,
of Denver, Colo.

Professor of Diseases of the Eye and Ear in Gross Medical College.

THE intimate association, physiologic and pathologic, of the eye and the brain has been overlooked to a very great extent by nearly all writers on ophthalmology, and to a less degree by the authors of works on diseases of the brain and nervous system. Reasoning from the close nervous connection existing between eye and brain, we are at once impressed with the clinical importance of the interdependence of these two wonderfully sensitive organs. In the course of a somewhat extensive ophthalmic practice during the past few years, I have met with a considerable number of cases which to my mind demonstrated vividly that this relationship should be better understood. A desire to acquire a clearer understanding of the subject myself, and possibly to bring out a discussion, was the incentive which prompted me to write this paper.

Even as late as 1859, the great ophthalmologist and scientist, Donders, who was then at his zenith in the development of optics as regards the laws of refraction and accommodation, brought to light a term hitherto unknown in its real significance—astigmatism; a word whose meaning our school children are now being taught. When this condition, astigmatism, is present only to a limited degree, it may cause what has been called for ages supra-orbital neuralgia. Viewing the matter just in a cursory way, do we understand how this pain is produced? Astigmatism, simply defined, is a congenital deformity, a lack of symmetry in the form of the eyeball. Do we mean to say, regarding the pain in this

[1] Read before the Colorado State Medical Association, June 18, 1895.

region, that the eye itself is in pain? By no means. The eye is in a fair state of health, with total absence of any inflammatory condition. We must say then that there has been communicated to the brain, the great mother of the nervous system, the fact that her dearest child is unable to do good work in refracting rays of light upon the fundus oculi, and this good mother is in distress on account of her child's deformity. But when the astigmatism is of a severe and complicated type there results an inflammatory condition which causes decided disturbance of the functions of the eye regarding sight. For instance, if the highly astigmatic eye cannot abstain from trying to perform labor for which it is not adapted and which is so severe in its effects on its mother, the brain, she must of necessity resort to some means by which the eye shall be given rest. Thus an inflammatory condition is set up by the brain withdrawing her power of vital resistance, and the impending forces at once attack this eye and it becomes congested, not only in its ocular and palpebral surfaces, but the deeper recesses and the essential mechanism of the organ, such as the ciliary process and the retina, are involved. Then the orbicularis muscle is commanded to shut off the light from the inflamed eye, and by these means the organ of vision is put in a comparative state of rest. Do we understand that the case portrayed is solely and distinctively an eye trouble? We do not; its cause originated at a given point, and, like a telegraphic message, word has been sent to the brain that the eye was in trouble; and, after deliberating over this complicated condition, the brain decided it was best to take the course described. By this assault upon the structures of the eye, supra-orbital neuralgia is relieved or prevented, and the inflammatory condition of the organ on the outside of the brain has been placed in its stead; by this means the seat of power has been protected and one of her branches only suffers. How interesting it is to reason on these conditions and to learn how nature protects her interests at every conceivable point, thereby preserving her pristine vitality. The beautiful provision of nature in the case stated is this: While the eyes only are impaired, the other parts of the system may be in good working order, performing all their functions without difficulty. But if the central irritation should have been continued, an inflammatory state of the seat of the nervous system would certainly have resulted; and this possibly might have terminated in a degenerative process to whose deadly influence, sooner or later, the brain would have been forced to succumb.

Optic neuritis is another well-defined disease of the eye which, in a majority of instances, points to brain complication. If a patient is suffering with continuous headache in optic neuritis, it is always indicative of serious disease, and if you can exclude three constitutional disorders, namely: anemia, kidney disease, and lead poisoning, and if, moreover, the headache is accompanied by vomiting, there is scarcely one chance in a hundred to fail in a diagnosis of a central lesion most probably tumor. The ophthalmoscope should never be neglected in cases of severe headache; it often leads us directly to a diagnosis.

Some time ago a lady was brought to me suffering with continuous headache which had been a source of annoyance for several months. Her vision was very much impaired; she read $\frac{3}{200}$ with one eye and $\frac{3}{200}$ with the other. Her vision had been failing continuously for six months. When I examined her with the ophthalmoscope I found a well-defined case of optic neuritis, the opacity of the disc being dense and striated. The only symptom which she had shown indicating disease prior to this eye trouble, was a slight unsteadiness of gait from which she claimed she had long since recovered. I suspected a central brain lesion, and so informed her attendant. I discouraged any treatment so far as the eye affection was concerned. She returned to her family physician. About a month later I was informed that she had succumbed to the disease and that a post-mortem showed a central brain tumor.

Optic atrophy, when not a sequel of neuritis, is generally a symptom of degenerative disease of the central nervous system. To undertake to classify all the different forms of atrophy is not necessary on this occasion; neither is it satisfactory, for there are so many exceptions that the rule is nullified in many cases. So, for convenience and to save time, I will merely mention the progressive forms as being especially destructive, whether from multiple sclerosis, locomotor ataxy, syphilis, or embolism of the central artery. Atrophy from pressure of a tumor on the nerve is comparatively uncommon, as only a small proportion of the intracranial tumors are situated in this vicinity. Hemorrhage by compression, exostosis, cheesy tubercular masses, neoplasms, and hydrocephalus internus, are all quite common causes of atrophy by pressure about the chiasm. In all cases of early atrophic disease of the nerve it is well to measure the amount of vision and ascertain if any great error of refraction is present. We invariably have diminution of color perception and a contracted field. In order to determine whether there is really disease of the optic

nerve or whether it is a central brain lesion, just observe the following rule: The reaction of the pupil to light is lessened in diseases of the nerve, but not in diseases of the brain.

Inequality of pupils, unless it has been present from childhood, is nearly always indicative of a lesion of some character along the optic tract. When not due to this cause it possibly has been brought about by paralysis of a branch of the third nerve, following a long siege of sickness—typhoid fever, scarlet fever, diphtheria and whooping cough all predisposing to this condition. If the pupillary inequality cannot be traced to one of these causes, it is a good idea to seek for intra-cranial complications, and don't forget in cases of this kind to test for color blindness and to measure the visual field, as we often have small spaces like little scotomata producing a break in the field of vision as well as deterioration of color perception.

Aneurism. We sometimes find optic neuritis appearing in this affection, but it is not usual unless the aneurism is near the optic nerve, for instance, in the internal carotid or anterior cerebral artery. Total loss of sight of one eye, which after a time extends to the other, causing complete blindness, often occurs without the slightest ocular disturbance save the constant failure of sight. In the beginning of a disturbance like this we frequently have a symptom known as hemianopsia, a permanent defect of half the visual field of both eyes from pressure on the tract, or chiasm; and sometimes from a functional disturbance of the optic centers in one cerebral hemisphere. The hemianopsia is usually either monolateral or bilateral, the latter variety affecting the external half of one field and the internal half of the other; the former the temporal or nasal half of each eye. Bi-nasal hemianopsia can exist only as a symmetrical disease of both optic nerves, pressure upon external part of each nerve. Bi-temporal hemianopsia, loss of the outer half of each field of vision, always means damage to the internal half of each nerve in front of the chiasm, and this would necessarily injure the fibers of each nasal half of the retina. The complete loss of one eye functionally and the other left undisturbed, signifies derangement of the nerve between the chiasm and the external part of the optic foramen. I think I have mentioned all the principal regional points which are requisite to properly understand the several forms of lack of visual acuity due to lesions along the optic tracts. In hemianopsia the color sense is usually found to be perfect in the unaffected half of the visual field. Paralysis of the third nerve, without impairment of sight, is gener-

ally produced by aneurism of the posterior cerebral and communicating arteries.

For want of time, many diseases have been omitted which have more or less bearing upon this rare field. I have tried to discuss those only which are complicated the most often with brain trouble. Concussion, compression, and laceration of the brain involve the eye oftentimes, and in many instances the *ophthalmoscope especially*, and also the perimeter, aids very materially in arriving at a correct and satisfactory diagnosis as to the graveness of the injury.

A CASE OF GUNSHOT INJURY INVOLVING BOTH EYES, STUDIED NINE YEARS AFTER THE ACCIDENT.

By Robert R. Saunders, M. D.,

CLINICAL ASSISTANT IN THE OPHTHALMOLOGICAL DEPARTMENT OF THE JEFFERSON MEDICAL COLLEGE HOSPITAL.

THE following case presents certain features of sufficient interest for brief record:

J. H., a man 21 years of age, presented himself for treatment on June 16, 1895, and gave the following history: On September 26, 1886, he was accidentally shot by a companion. Bird shot entered his right arm, chest and face, and one shot was found imbedded in the sclera of the right eye near the outer canthus, while another shot was removed from the same eye above the inner canthus.

At that time he was under the care of Dr. Charles Schaffner, who very kindly has permitted me to incorporate his notes of the case:

"October 7, 1886, J. H., 13 years of age, accidentally shot in both eyes twelve days ago.

"O. D. vision = $\frac{5}{15}$; J. 8, from 10 to 50 inches. The ophthalmoscope reveals a discolored and congested optic disc; many streaks of lymph and small clots, together with glistening spots of choroidal change in outer half of the fundus oculi, and a limited separation of the retina in the lower portion, together with a hemorrhage in the vitreous. A small shot was found sticking in the right eye near the outer canthus.

"O. S. vision equals light perception. A shot had penetrated the lower and inner border of the cornea, and is probably located in the vitreous, although examination of the deeper structures is impossible, owing to the traumatic cataract which is present. The tension is diminished."

Under Dr. Shaffner's treatment, the right eye gradually improved, and one month later the vision was $\frac{20}{30}$. By January 4, 1887, the vision had risen to $\frac{20}{20}$, and he was able to read the finest print, from $3^1/_2$ to 11 inches.

The ophthalmoscope at that time revealed slight clouding of the optic disc, and a greenish white scar in the lower floor of the eye, extending in the form of a streak from the far periphery of the eye-ground almost to the optic disc. The left eye had in the mean time passed into a condition of atrophy. Enucleation was advised, after consultation with Dr. Strawbridge, but declined. The shot was removed from the sclera of the right eye.

The patient has suffered no inconvenience since the accident to the present time, and comes now for correction of his refractive error.

On studying the right eye under thorough mydriasis, the following interesting lesions were discovered:

The media are clear, the disc is nearly round, its edges are hazy and the scleral ring is broadened to the nasal side. The veins are rather full and there is slight infiltration of the arterial lymph sheaths. The macula is represented by an oval, ring-shaped area, with a crescent below it, and beyond this a patch of shifting reflex. On the lower temporal artery, as it curves to the macula, there is a somewhat conical patch of lymph. Two and one-half diameters from the inferior border of the nerve-head begins a broad rent in the choroid and retina, which widens out towards its center and then becomes narrow as it extends forward; it extends as far forward as ophthalmoscopic examination is possible. The floor of this rent is composed of glistening-white, exposed sclera, and its edges are bordered with streaks of pigment. At its upper end there is a prolongation in which the choroid has not been absorbed and across which the larger choroidal vessels can be seen. Up and out from the disc, in the far periphery of the eye-ground, there is a patch of atrophy, the lower border of which begins in a mass of pigment. The atrophic area, expanding into a fan-shaped extension, can be followed as far upward as ophthalmoscopic examination is possible.

The tension of the left eye is diminished, but the eye is not tender on pressure. The iris is incarcerated in a scar situated at the lower and inner corneal limbus, and the pupil extends as a slit up and out from this point, and is bound down at all points to the capsule of the lens, the iris tissue itself bulging forward in the form of the so-called iris bombè. The anterior chamber is practically obliterated. Through the pupil remains of the cataractous lens are discernable,

There seems very little doubt, both from the clinical history kindly furnished by Dr. Shaffner, and also from present ophthalmoscopic examination that a shot entered the right eye below the

cornea, penetrated the sclera, choroid and retina, passed across the vitreous and emerged at a point up and out from the disc, the wound of entrance, so far as the fundus oculi is concerned, being now marked by the lower patch of atrophy, and the wound of exit by the fan-shaped area situated in the upper portion of the eye-ground. With the exception of these lesions, so situated as not to interfere with direct vision, there are only a few remnants of the former choroiditis which existed and is described in Dr. Shaffner's notes, namely, the patches of lymph and slight changes in the pigment below the macula.

Careful search in the sclera fails to reveal with certainty any scar indicating the point of entrance of the shot or its exit, but as the shot which were known to exist there and were extracted by Dr. Shaffner have also left no scars, this is not a remarkable circumstance.

It is interesting to note that although the pathologic lesions obtain in the left eye which are not infrequently followed by sympathetic irritation, or even inflammation, no such signs have ever occurred.

So far as the right eye is concerned, the case may be relegated to those in which an unusual traumatism has been followed by complete restoration of the visual functions, and is particularly interesting from the ophthalmoscopic standpoint, inasmuch as the present lesions can be compared with those studied by Dr. Shaffner a few days after the injury, nearly nine years ago.

A CASE OF RETENTION OF A METALLIC SPLINTER IN A BLIND EYE FOR SEVENTEEN YEARS WITHOUT THE OCCURRENCE OF SYMPATHETIC INFLAMMATION.

A CASE OF MEMBRANA PUPILLARIS PERSEVERANS IN BOTH EYES IN AN ADULT.

By J. M. Banister, A. B., M. D.,
of Fort Leavenworth, Kan.

Captain Medical Department, United States Army.

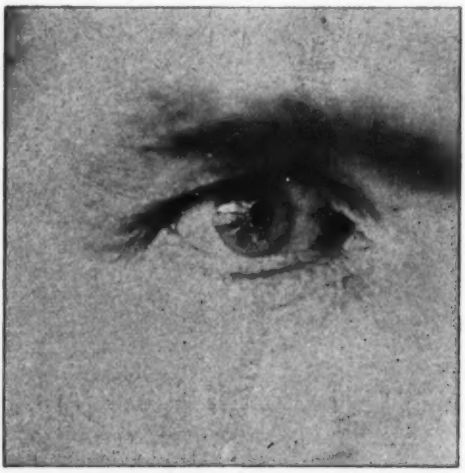
Mrs. M., 19 years of age, of Leavenworth, Kan., lost her left eye seventeen years ago, under the following circumstances: While watching a little playmate, who was engaged in exploding percussion caps by placing them upon a piece of iron and striking them with a hammer, something struck the cornea between the open lids, and opened the anterior chamber. The mother states that she saw the eye immediately, and that when she pulled the lids apart a piece of cap fell out from between them. This assertion on the part of the mother caused the family physician, to whom she took the child immediately after the injury, to conclude that nothing had penetrated into the interior of the globe; that, in short, the piece of cap, which the mother observed, had simply wounded the cornea, and had fallen out when the lids were opened. The eye was speedily lost, but without any very severe suffering, if the mother gives a correct version. From that time, until very recently, the blind eye caused no inconvenience whatever. In November, 1894, however, the good eye began to exhibit symptoms of sympathetic irritability, which symptoms increased in severity until March, 1895, when the patient consulted me with reference to her case. Upon examination I found vision in the right eye normal, tension normal, and no evidence of inflammation of the iris, or deeper structures, though the organ was very irritable. The injured eye was absolutely blind, tension subnormal, globe somewhat atrophied, cornea staphylomatous and exhibiting yellowish plaques in certain local-

ities. The iris seemed to be applied to the membrane of Descemet. Believing that an attack of sympathetic irido-cyclitis was imminent, I advised the speedy removal of the blind eye. On March 27 the latter was enucleated. The following day I opened the removed organ in the presence of Assistant Surgeon W. F. Lippitt, U. S. A., and found near the periphery of the iris, in the equatorial plane of the globe, a piece of iron, or steel, about 3 *mm.* in length and about 1 *mm.* in thickness wedged in between the iris and lens, and surrounded by a deeply pigmented capsule of iris tissue and inflammatory exudate. The iris thoughout the rest of its extent was atrophied, and firmly attached to the membrane of Descemet, the anterior chamber being thus obliterated. The lens was cataractous, being of the color and consistence of cheese, and was pushed forwards toward the anterior pole of the eye. The vitreous was of the same color, and semi-solid. The choroid, ciliary processes, and retina, were thoroughly atrophied. As before mentioned, the foreign body was a fragment of iron, or steel, and not a piece of cap. It must have been a splinter from the hammer, with which the caps were exploded, or a fragment from the piece of iron upon which they were placed. This splinter had evidently passed through the cornea and iris, and had been entangled in the posterior portion of the iris, where it had remained for *seventeen years*. The eye had been lost by an irido-cyclitis, and not by a panophthalmitis. Although cases are on record in which foreign bodies have remained in the eyes for many years without causing sympathetic trouble in the fellow organs, yet those in which a fragment of oxidizable metal has been embedded in such an intolerant tissue as that of the iris of an eye, lost through a traumatic irido-cyclitis, for the length of time involved in the present instance, must be *unusual*, to say the least. This consideration induces me to place this case upon record. Since the removal of the offending eye, in the case just reported, all irritability of the sound organ has entirely disappeared.

A CASE OF MEMBRANA PUPILLARIS PERSEVERANS IN BOTH EYES IN AN ADULT.

Recently the writer examined the eyes of Joseph Macho, 47 years of age, of Fort Leavenworth, Kan., and found in each eye a *persistent pupillary membrane.* This condition was more marked in the right eye than in the left, though very evident in the latter.

Vision in the right eye was very defective, being only $\frac{20}{200}$, examination with the ophthalmoscope revealing cloudiness of the vitreous body, and the presence of several large floating membranous opacities in the same, to which cause the poor vision should undoubtedly be accredited. Vision in the left eye was nearly normal, being $\frac{20}{25}$. The ophthalmoscope revealed nothing abnormal in this eye, beyond the pupillary membrane. The accompanying illustration, from a photograph taken in my presence, gives a representation of the appearance of the "membrane" in the case of the *right eye*. The left eye presented a very similar appearance. The fibers constituting the so-called membrane were not attached to the cornea or lens, though upon casual examination they presented an appearance very similar to that



usually observed in the case of anterior synechiæ. By oblique examination and the ophthalmoscope their true character could be easily determined. Under the influence of a mydriatic the pupils were dilatable, and perfectly regular in outline. In the illustration the outer margin of the pupil, and the fibers of the membrane in this situation, are obscured by the light reflex from the cornea. The fibers constituting the membrane pass over the outer margin, and the outer portions of the upper and lower borders of the pupil, and are united within the area of the latter in a kind of membranous expansion. Only the inner and central portions of the pupil are shown in the illustration, the membrane and light reflex obscuring the remainder.

ABSTRACTS FROM AMERICAN AND ENGLISH OPHTHALMIC JOURNALS.

By Charles H. May, M. D.,
of New York.

THE PARALLAX TEST FOR HETEROPHORIA.

Dr. Alex. Duane, New York, (*Archives of Ophthalmology*, April, 1895). Eight years' use of this test, upon a large number of patients, has convinced Dr. Duane of its utility, and he recommends it as almost universally applicable, precise and accurate. The method of using the test is as follows:

"The patient is placed in the primary position with head erect and eyes directed straight forward or slightly below the horizontal plane (this latter especially in making the test for near points). The object of fixation, which should be twenty feet distant, may be a candle-flame, but preferably is a white spot 1 to 2 *cm.* in diameter, upon a dull black surface of some considerable extent. By this arrangement all danger of projecting the image upon a surface beyond is done away with, and the chance of a confusion with surrounding objects is prevented. The patient's gaze being directly fixed at the spot, a card is placed before one eye and passed alternately from that to the other, the patient being at the same time asked whether the spot appears to move, and, if so, in what direction. If it remains perfectly stationary there can have been no deviation behind the card, and the position of fixation of both eyes is perfect. If, however, the spot moves, it must occupy a different position as seen by the two eyes; *i. e.*, there is really a diplopia present which our method of observation has unmasked. Thus, if on uncovering the left eye, the object (which was previously seen by the right eye and is now seen by the left) appears to move to the patient's left, there is really a homonymous

diplopia (homonymous parallax) which differs from ordinary diplopia only in the fact that the two images are seen alternately instead of at the same time; if the object seems to move to the right there is crossed diplopia (crossed parallax); if the object moves down, the eye must have been higher behind the screen (left hyperphoria, left parallax); if the object moves up, the left eye must have been lower behind the screen (right hyperphoria, right parallax). In order to determine the amount of this alternate diplopia we place prisms of the appropriate direction and strength before one eye until the movement is abolished. Thus, supposing that when the left eye was uncovered the object seemed to move down and to the left (homonymous and left parallax, indicating a condition of hyperesophoria), two prisms are placed before this eye with their bases respectively, down and out, and increased in strength until the movement has become *nil*. The strength of the prism having its base down will measure the degree of hyperphoria, and that of the prism having its base out will indicate the degree of esophoria present. For near points the test is made in the same way, a small dot on a rather large card being employed, and the movement of the dot upon the card (and not of the card itself projected against some distant object) being observed."

Regarding precision, he states that a prism of $^1/_2{}^\circ$ is sufficient to neutralize or to produce a decided lateral parallax; "and a vertical movement corrected by a prism of even $^1/_8{}^\circ$ is clearly noticeable. This precision makes it useful in determining whether a glass that the patient is wearing has or has not a prismatic element, and whether it is centered or not. If, for instance, a patient who has no hyperphoria, shows, with his glass on, a vertical parallax, it proves either that the glass has a prismatic element in it or is faultily adjusted (decentered) so as to produce a prismatic effect."

In regard to accuracy, he claims it is one of the most reliable of all the different methods and that it is sometimes preferable to other methods. In all the other methods in which prisms are used either in trial frames or in phorometers, the patient often unconsciously tries to bring the two images in line and thus conceals a certain amount of the insufficiency which exists. Even when the images are made to appear different (Maddox rod and Steven's spherical lens with small aperture), the impulse towards bringing the two dissimilar images into line is still present. In the parallax test the two images being seen alternately, there is not the same impulse to force them together.

EPHEDRIN—HOMATROPIN, THE NEW MYDRIATIC. A REVIEW OF THE WORK OF DR. GROENOUW AND DR. GEPPERT.

George F. Suker, M. D., Toledo, Ohio, (*New York Medical Journal*, June 8, 1895). The writer speaks very highly of this combination and considers it an ideal mydriatic for diagnostic purposes. It has been extensively used in the following form:

Ephedrin hydrochlor	1.00
Homatropin hydrochlor	0.01
Aq. destill.	10.00

This forms a clear, colorless solution, the instillation of which produces no irritation or only a slight smarting. It does not influence accommodation. The mydriatic action is rapid and powerful. After a single instillation the pupil begins to dilate in about eight minutes and attains a maximum in half an hour. After an hour the pupil begins to contract slowly and regains its normal size in from four to six hours; when dilated to the maximum, the pupil measures from 5 to 6 *mm*. This combined solution does not deteriorate as rapidly as homatropin, showing no signs of loss of action after three months. From his observation he concludes that "we possess in the ephedrin-homatropin solution practically an ideal mydriatic for diagnostic purposes, being rapid in its action, sufficiently intense, and of very short duration. Merck prepares this solution under the name of 'mydrin.'"

CLINICAL AND ANATOMICAL STUDY OF SEROUS OR SIMPLE CYSTS OF THE CONJUNCTIVA.

Dr. G. Rombolotti, Pavia, (*Annales d'Oculistique*, English Edition, March, 1895). The writer makes the following division of serous or simple cysts of the conjunctiva:

"*A first variety* is composed of those which are situated on the bulbar conjunctiva, and which are of congenital as well as of spontaneous origin. Saemisch says that these cysts are generally situated near the corneal margin; that usually they are not movable under the mucous membrane, which latter enters into the composition of their walls. They are round in form, with a thin, delicate wall of almost perfect transparency, allowing the contents, clear as water, to be seen.

"*A second variety* which has its seat on the bulbar conjunctiva near the corneal margin, and with almost the same morphological characteristics as those of the first variety, is composed of those which may be traced to a traumatic origin.

"*A third variety* of sub-conjunctival cysts includes those which are developed in the bulbar conjunctiva from dilatation of the lymphatic vessels of the mucous membrane. They are generally small like a small transparent worm, or disposed in groups.

"*A fourth variety* of cysts which deserves special classification is formed of those which are developed in the reflected portion of the conjunctiva; * * * it appears that their development is due to a cystic dilatation of the acino-tubular glands of Krause."

DIABETIC RETINITIS.

Oscar Dodd, M. D., Chicago, (*Archives of Ophthalmology*, April, 1895). The writer calls attention to the fact that "although the existence of diabetic retinitis has been known for a long time, the literature on the subject is very meager and unsatisfactory." He has tabulated the histories of forty-seven cases, all the cases of pure diabetic retinitis reported in literature, and from the study of these he has collected characteristics indicative of the disease. He mentions the authors who made the most important contributions to the subject and then considers the ideas of different writers.

"The *age* at which diabetic retinitis occurs is generally an advanced one, most of the cases occurring after 45."

The writer mentions several *types* of retinal changes, considering that form the most frequent in which there are "small glistening spots of degeneration interspersed with minute hemorrhages principally in the central part of the retina." These spots are small, rarely exceeding $^1/_4$ to $^1/_2$ the size of the optic disc, vary in shape, being usually nodular and irregular, and seldom stellate around the macula as in albuminuric retinitis; in color they vary from a dull to a glistening white or yellowish-white. In a second class of cases, the whitish patches and hemorrhages are scattered over the whole fundus.

"Edema and *active changes* in the retina and optic nerve are rarely present although occurring so frequently in albuminuric retinitis. The vessels are usually of normal size and contour. This is one of the chief distinguishing features between diabetic and albuminuric retinitis."

"The frequency of opacities and hemorrhages in the vitreous has not proved so great as would be supposed from literature, several authors mentioning it as a characteristic of the disease. It was present only seven times among the forty-seven cases reported."

"The *subjective symptoms* of this disease are very slight in the cases with minute hemorrhages and punctate spots in the center of the retina. The retinitis may be accompanied or preceded by the diabetic amblyopia, which, according to Bouchardot is present in one-fifth of all diabetic cases, and frequently occurs without any fundus change. Most of the cases complain only of a dimness of vision, especially for near work, and are seen only upon their applying for glasses to correct the defect. There may also be a sensitiveness of the eyes to light and a flickering before the eyes at an early stage of the trouble, due to an irritation of the retina. When the change affects the macula itself or there are large hemorrhages into the vitreous, the visual disturbance is profound.

"Of the *conditions following diabetic retinitis* the most frequent is atrophy of the optic nerve."

Concerning the *pathology* of the ocular changes he gives a report of the few examinations which have been made and then says: "We see from these cases that the important changes are in the blood vessels. These affect principally the small vessels, and are not so extensive as in albuminuric retinitis. The partial closure of the vessels tends to rupture, producing the small hemorrhages which are so common in this disease, and predominate over the degeneration which is the characteristic change in albuminuric retinitis. If there is anything in the pathology of the disease distinguishing it from other forms of retinitis it is the peculiar changes taking place in the small arteries and capillaries. In other forms of retinitis we have changes in the blood vessels, but they are equally as great or predominate in the large vessels."

In discussing the *differential diagnosis*, which is one of the principal objects of the paper, he gives the table prepared by Badal:

DIABETIC RETINITIS.	ALBUMINURIC RETINITIS.
1. Marked tendency to atrophy of the optic nerve.	1. Less tendency to atrophy which occurs especially at a late stage.
2. Multiple hemorrhages, round and disseminated.	2. Hemorrhages equally multiple, but elongated, and occupy especially the posterior layers.
3. Alterations diffuse.	3. Affect principally the circumpapillary and macular regions.
4. Apoplexies do not last to a late stage.	4. Last throughout life.
5. Small disseminated spots with some exudate.	5. Whitish fatty spots with infiltrations.
6. Color sense, *nil*.	6. Persists a long time.

From a study of the collection of cases, Dodd tabulates the distinguishing features as follows:

DIABETIC RETINITIS.	ALBUMINURIC RETINITIS.
1. Groups of bright glancing spots in retina, irregular in outline, usually in central part, but frequently affecting whole of fundus.	1. At first a group of bright bluish-white spots in center of retina, often forming stellate patch about the macula.
2. If the spots are larger there still exist small dots and lines, and they never run together.	2. Spots may run together and involve all of central part of retina.
3. Arteries and veins not much changed in appearance.	3. Arteries narrowed, veins large and irregular.
4. Optic nerve either not affected or atrophic.	4 Optic nerve swollen, and outline indistinct.
5. Retina not diffusely affected.	5. Retina infiltrated.

DISEASES OF THE EYE DEPENDENT ON THE GRIP.

Thomas R. Pooley, M. D., New York, (*Am. Medico-Surg. Bulletin* report of the March meeting of the New York Academy of Medicine, April 15, 1895, and *Amer. Journal of Ophthal.*, May, 1895). After mentioning the many diseases of the eye which have been attributed to the grip by various authors, the writer says: "After a careful review of this subject, I would state the following as my conclusions: (1) That eye affections following the grip are comparatively rare. (2) That many of the cases reported as being due to grip are fanciful, and need more substantial proof. (3) Grip may affect the eye by a direct inflammatory process, or by extension from the accessory sinuses. (4) It may affect the nerves of the eye. (5) It is especially liable to affect the conjuctiva, the uveal tract, and the tissues of the orbit, and, perhaps, the fibrous capsule. (6) In some of these cases the extension is by metastasis and in others by direct continuity. (7) Before attributing any eye complication to the grip, careful and thorough scrutiny is necessary to exclude other causes, such as syphilis, alcohol, etc.

A NEW OPERATION FOR PTOSIS.

Dr. Mules, London, (Report of the May meeting of the Ophthalmological Society, *The Lancet*, May 11, 1895). Dr. Mules brought before the society a new operation for ptosis. It was first described by him at the last international Congress of Ophthalmology at Edinburgh in August, 1894. He now gave the results of his

further experience of its effect. The principle of the operation was to substitute the frontalis muscle for the levator palpebræ by extending the former muscle to the margin of the lid by a permanent wire suture. Two needles with eyes near their points were passed deeply through the frontalis tendon over the eyebrow, and their points brought out at the margin of the lid behind the lashes, taking up a substantial part of the tarsal cartilage on their way. A piece of silver wire was threaded through each needle, which was then withdrawn, leaving the loop of wire passing from the brow to the edge of the lid and back to the brow again. This was then tightened until the lid was sufficiently raised, the edge of the lid being slightly grooved by an incision to allow the wire to sink into the substance of the lid. One end of the wire was then passed under the skin and made to emerge by the side of the other end of the wire. The two ends of the wire were then twisted in each other until the lid was wired permanently, the ends cut off, and the wire allowed to sink below the level of the skin. The skin at this point and at the lid margin healed over the wire, which remained permanently fixed in the substance of the lid. From further experience it was found that the wire remained in position without causing irritation; the lids could be closed and remain closed during sleep. All kinds of wire had been tried, but it had been found that silver wire was the most satisfactory. It was necessary to note at the time of the operation the situation of the twisted end of the wire in case it became necessary to remove the suture afterwards.

INTERSTITIAL KERATITIS AND SYNOVITIS WITH A REPORT OF A CASE IN WHICH BOTH WERE UNILATERAL.

G. Crawford Thomson, M. D., Durham (*The Lancet*, April 13, 1895). The writer reviews the literature of this subject and shows that credit is due to Foerster, who in 1877, "first observed the close clinical connection between the two diseases, and for having first pointed out the non-rheumatic character of the joint disease." He then describes in detail a case in which "both affections remained unilateral" a rare exception to the law formulated by Hutchinson that "interstitial keratitis in its typical form is always in the end symmetrical." * * * "As to the causation of both affections, I do not feel justified in attributing them to inherited syphilis simply for the fact that interstitial keratitis existed together with knee-joint affection. The history of my case, complete as it is, contains scarcely anything to suggest syphilis."

"Complete recovery took place under a simple tonic treatment, neither mercury nor iodid being given. In a disease with a decided tendency to recovery, this latter fact is not of much value; still it in some respects corroborates the view taken here."

The writer argues against the exclusively specific nature of interstitial keratitis, doubting the correctness of the rule laid down by Hutchinson: "Interstitial keratitis in its typical form is always a consequence of syphilis and in itself sufficient for the diagnosis." The results of most other ophthalmologists, as shown by a recent table of Ogilvie, do not bear out Hutchinson's rule. "Another argument against the exclusively specific nature of interstitial keratitis given by Ogilvie is that it has been observed in dogs, and experimentally produced in rabbits. Since the publication of his paper several cases of interstitial keratitis have been observed in bears by Hennicke, and the diagnosis has been confirmed by microscopical examination."

"According to Fournier four theories exist concerning the causation of interstitial keratitis: (1) That it is a cachectic malady—'une manifestation de la misère organique' (Panas); (2) that it is produced by scrophulous or strumous disease (W. Mackenzie); (3) that it is exclusively, or nearly exclusively, due to syphilis (Hutchinson); and (4) that it is a lesion of natural nutrition (Fournier). He dismisses (2) with little comment as no connection with strumous disease could ever be proved; he considers (3) to be contrary to the clinical facts as interstitial keratitis is met with apart from any specific influence; and refutes (1) because interstitial keratitis is met with in subjects otherwise apparently healthy, and in private as well as in hospital practice. I fail to see that Fournier's explanation differs materially from that of Panas." A lesion of general nutrition, "cachexia" and "organic misery" are only different expressions for the same condition, or for different degrees of the same condition. Therefore, the fact that interstitial keratitis is met with in apparently healthy subjects is explicable by either theory only in one and the same way, *viz*: that the defective general condition in these cases is apparent as localized or limited to one special organ. This same view has been quite recently taken by Panas who admits that between Fournier's dycrasia and his cachexia practically no difference exists, and that he therefore willingly accepts Fournier's denomination instead of his. From the above the conclusion has to be drawn that in the production of interstitial keratitis hereditary syphilis is probably operative in between 60 and 70 per cent.

"I, therefore, from my own case, as well as from the experience of Arlt and Lavergne, draw the following conclusions: Neither interstitial keratitis nor the synovitis nor their coexistence is itself absolute proof of hereditary syphilis; that the joint disease also is due a general defect of nutrition, in the production of which inherited syphilis plays a prominent part, the extent of which we are not able to give in figures. In what this disturbance of general nutrition consists, what general condition constitutes the connecting link between interstitial keratitis and synovitis, is merely a matter of conjecture."

A NEW APPLICATION OF THE KERATOMETER OF JAVAL.

Dr. W. F. Southard, San Francisco, Cal., (*Medical Record*, April 6, 1895). Dr. Southard calls attention to the fact that Javal's ophthalmometer may be used to obtain an excellent view of the cornea and iris. "Seating my patient before this instrument and lighting but one of the lamps, the light was made to fall directly upon the cornea. After a little maneuvering with the telescope a fairly good view of the iris was obtained. As soon as the iris came into the field the reflexes of the disc appeared. Further experimenting brought out the fact that to see these surfaces distinctly it was necessary to take the illumination from a little back and to the same side as the eye to be examined, then focus the light upon the eye with a mirror of eleven-inch focus. I used a gas flame for illumination, and my head-mirror which I held in my hand. The details of the cornea, if opacities are present are very distinctly seen; the color of the iris, its structures, the crypts, contraction furrows, pigment patches, and papillary zone are all clearly brought out. Opacities on the anterior surface of the lens may be seen. The image thus obtained is magnified and all the details easily made out, such as tumors or synechiæ. By mounting the mirror on a stand both hands are free to make a sketch of the actual situation of adhesions. While we are measuring the curvature of the cornea it takes but a moment to carry out this examination should it be thought best to examine the iris. If the patient will look through the opening in the disc, thus relaxing the accommodation, the pupil will dilate and uncover any adhesions of iris should they be present. A slight disadvantage in examination by this method is due to double images of the large disc and wires. By revolving the tube, the image caused by the prism which lies just outside the optic axis of the tube will be carried around the stationary image, thus all portions of the iris will be seen in turn.

PECULIAR PERVERSION OF THE COLOR PERCEPTION.

Homer E. Smith, M. D., Norwich, N. Y., (*Medical Record*, March 9, 1895). Dr. Smith reports the case of a boy of 11 years of age in whom, among other evidences of functional disturbances referable to the nervous system there was a peculiar perversion of the color perception. The following is the portion of the report which relates to the eyes: "Vision, O. D. $= \frac{4}{20}$; O. S. $= \frac{6}{20}$. There is no strabismus or conjugate deviation of the eyes. Motion perfect in all directions. Pupils equal and of normal size, contract to light exposure, consensually, to convergence, and to peripheral excitation of the retina (Wernicke's test). Fields, marked peripheral limitation, greater in right. No scotomata. Accommodation not impaired. Selects colors correctly as to shade, but each appears in its complementary, *i. e.*, red is called green, and green appears as red. Blue looks like yellow, and yellow, blue. White appears brown. Has micropsia and metamorphopsia. There is moderate photophobia present. Has in each eye monocular polyopia. The ophthalmoscope shows O. D. diffuse neuro-retinitis. The bulk of the trouble is in the retina, which is edematous and opaque. The papilla is not swollen, but is markedly congested with a nearly complete obscuration of the choroidal ring. Veins slightly tortuous, no hemorrhages. O. S. same condition, lesser degree." The child was given iodid of potassium and tonics, and after four weeks all these symptoms had disappeared. The writer thinks that most likely the lesion was an intra-cerebral effusion, probably hemorrhagic, situated in the left optic thalamus with pressure effects involving the internal capsule in the thalamo-lenticular division. (Dr. Smith sent a number of reprints to various prominent oculists of the United States inviting an opinion regarding the diagnosis in this case. A number of different views were expressed in the answers which he received; most of them regarded the case as an example of hysteria. C. H. M.)

BLOOD-STAINING OF THE CORNEA.

Mr. Treacher Collins, London, (Report of the May meeting of the Ophthalmological Society, *The Lancet*, May 11, 1895). Mr. Collins found that this staining of the cornea, which was of a greenish or reddish-brown color, was due to the presence of a number of highly refracting granules scattered thoughout its substance. These granules are not located with any definite relation

between laminæ of the fibrous tissue; they agreed in their spectroscopical appearances and chemical reactions with hematoidin. He found that in some of the cases associated with granules of hematoidin there was a substance which gave iron reaction with ammonium sulphid, and which was probably hemosiderin. In eyes in which this discoloration occurred the tension was generally increased, the exit of fluid through the angle of the anterior chamber being obstructed by the accumulation of blood clots. He was of opinion that at first hemoglobin passed into the cornea from the anterior chamber through Descemet's membrane, and that the hematoidin, which is insoluable in the fluids of the cornea, was then precipitated there. The whole of the cornea was at first affected, and when this was the case the condition could not be distinguished from that in which blood-clots completely filled the anterior chamber. The absorption of the hematoidin granules commenced at the periphery equally in all directions, so that by degrees a narrow ring of clear cornea appeared around the stained area. The appearances then presented were strikingly similar to those of a lens dislocated into the anterior chamber. The absorption of granules becomes slower and slower the further they are removed from the sclero-corneal margin. He had seen one case in which the discoloration had completely disappeared in the course of about two years.

ABSTRACTS FROM FOREIGN OPHTHALMIC JOURNALS.

By Casey A. Wood, M. D.,
of Chicago.

VARIATIONS IN THE PUPIL ACCOMPANYING PULMONARY TUBERCULOSIS — THE OPERATIVE TREATMENT OF HIGH DEGREES OF MYOPIA — MYOMA OF THE CHOROID — THE VOSSIUS METHOD IN CATARACT EXTRACTION — REPAIR OF A LARGE RUPTURE OF THE ANTERIOR LENS CAPSULE WITHOUT THE FORMATION OF CATARACT — GLAND-LIKE BODIES BENEATH THE ANTERIOR CENTICULAR CATARACT.

VARIATIONS IN THE PUPIL ACCOMPANYING PULMONARY TUBERCULOSIS.

In this article Rampoldi[1] reviews the opinions of several writers on the subject and publishes his own experience. At the last International Medical Congress Destrée[2] read a paper in which he claimed that in 97% of cases of tubercular phthisis he observed an unequal dilatation of the pupils dependent upon irritation of the sympathetic plexus at the hilus of the lung from disease in the bronchial glands. This sign, he claims, often precedes the invasion of the lung tissue and is a sure indication of tuberculosis of the bronchial glands. Cardarelli draws attention to the fact that the tubercular character of swelling in the peribronchial glands has been recognized since the earliest times, and that these glands, like the mesenteric, may retain the bacillus tuberculosis in a state of latency. Destrée later affirmed that after long continued and daily study of these cases he was able to state positively that the pupillary condition is the result of swelling of the peribronchial glands,

[1] R. Rampoldi: Ancora sulle variazioni pupillari dipendenti da malattie polmonari di natura tubercolare. *Annali di Ottalmologia*, Anno XXIII., Fasc. 6.

[2] Un segno premonitorio della tubercolosi polmonare. *Riforma Medica*, Anno X., No. 79.

which, pressing upon the filaments of the sympathetic, brings about the dilatation referred to, and that he had confirmed the fact of the pressure upon the nerve by many autopsies. Moreover, recent researches have proved that the peribronchial glands are usually infected very early in the disease, probably the first tissue invaded, and if we could be put into possession of a sign that would indicate that invasion, it is easily understood how important it would be from the standpoint both of diagnosis and treatment.

Rampoldi shows that he was the first (in 1885) to draw attention to this sign of pulmonary disease. Later (in 1886) he published a case which seemed to confirm the experience of Oehl "that it is possible to transmit a primary excitation of the vagus to the pupil by way of that sympathetic branch which runs from the superior cervical ganglion to the vagus itself."

In addition to this sign the author believes the following history to furnish evidence of further implication of the ocular nerve supply by tubercular disease of the lungs:

R. A., domestic, 16 years of age, seems in good health, but has suffered for three years with a slight cough, thought to be bronchitic. She visited the klinik on account of *a falling of the right upper lid* that had lasted the previous fortnight. A careful examination of the eyes was made and it was found that the patient had a decided ptosis on the right side, accompanied by a marked contraction of the corresponding pupil, which was sluggish to light and accommodation — in other words an unequal *dilatation* of the *two* pupils. There was no trace of posterior synechiæ, and no refractive error. Vision was normal both for distance and near.

Chiefly on account of the irregular innervation of the iris and lavator palpebræ superioris (not otherwise explained), Rampoldi suspected pulmonary disease and sent the patient to the medical klinik. She was found to have tuberculosis of the right apex.

THE OPERATIVE TREATMENT OF HIGH DEGREES OF MYOPIA.

This is an interesting paper contributed by Prof. Vossius[3], who describes very graphically the plague that threatens not only his own but other countries — that of shortsightedness. Not only is myopia on the increase, but it spares no class and no age, and we are without effective means of putting a stop to its onward march.

[3] A. Vossius: Ueber die operative Behandlung der Myopie nebst Bemerkungen ueber die Staroperation. *Beitraege zur Augenheilkunde*, XVIII. Heft. p. 49.

Owing to a diminution in the apparent size of distant objects seen through them, lenses which fully correct the refractive error do not give much relief, in the higher degrees of shortsightedness especially. Such patients content themselves either with a partial correction, which helps them very little, or they do not wear glasses at all—with the idea of conserving their sight—and thus remain practically blind so far as the outside world is concerned.

Many are the devices that have from time to time been tried with the purpose of radically removing the curse, the short sight. Purkinje, who suffered from a medium degree of myopia, was in the habit of placing on his eyes, over night, a small bag filled with half a pound of iron filings, and felt rewarded when he was able to read the numbers on the houses across the street next morning! Yet he did not succeed either in curing or lessening the amount of his refractive error.

The operative treatment is the only one to which we can look with any confidence for the permanent relief of the more distressing forms of myopia. Velpeau, in consequence of the belief that shortsightedness is the result of pressure upon the globe exerted by the external ocular muscles, advised section of certain of the latter. But this proceeding, as well as the plan of Galezowski (the removal of a crescentic portion of the cornea), soon fell into disuse.

In recent years removal of the lens has been more successful in lessening the amount of high degrees of myopia, in increasing the visual acuity, in improving the working capacity and in diminishing the ever present danger of further inroads on the part of the disease.

The first mention of treating excessive myopia by aphakia appears in Beer's book, edition of 1817. The suggestion was not acted upon until 1858, when Mooren brought the matter to the notice of the Heidelberg Congress.

Donders objected to the operation on account of the loss of accommodation and because he could not see how the fundus changes could be improved thereby.

In 1887, Fukala first presented his paper, with a report of two cases successfully operated upon, before the Vienna Medical Society, and since that time has frequently presented the claims of a method with which his name is very properly linked.

The following is a summary of Fukala's conclusions regarding this important matter:

1. The amount of myopia present before the operation determines, of course, the refractive state after removal of the lens. In any event the effect seems to be greater than that which we are accustomed to look for after extraction of senile cataract. In the latter case we usually find a 10 D. glass necessary to replace the cataractous lens, but Fukala found the optical value of an extraction in high myopia to vary from 12.75 to 20 D. This he thought to be due to the abnormal shape (and consequent effect) of the myopic lens upon the refraction.

2. The central visual acuity improves greatly as a result of the operation, and thus improvement may amount to four, five and even ten times the original.

3. The marked convergence of the visual axes and the abnormal bending of the head in near fixation are relieved — an important consideration in progressive myopia. When the myope, as the result of an operation, becomes a hypermetrope or an emmetrope, he must, of course, wear the glasses necessary to enable him to read at a desired distance. In this case the retinal images become measurably larger.

4. There is, of course, a loss of accommodative power [In his earliest paper Fukala claimed that some patients still retain a certain amount of accommodation after removal of the lens. C. A. W.] following operation, but this is not without its advantages since accommodative efforts are undoubtedly a frequent cause of the increase in the myopia.

5. When both eyes are affected by myopia of high degree, but unequally, it often happens, in consequence of the impossibility of binocular vision, that a divergent strabismus results. This often disappears after the operation.

It is in such cases, and indeed in most instances where binocular vision for either distance or near is absent (owing to the high degree of the myopia) that Fukala thinks the most useful results are obtained.

Fukala considers the following to be the indications for the operation: the absence of chorio-retinitis; adults having at least 14 or 15 D. of myopia; children under 10 years of age as soon as the myopia has reached 10 D., as these patients will, in all probability, acquire a higher degree later in life. In myopes of 17 D. one eye *may* be operated upon, although the removal of *both* lenses in such cases is indicated; in those of 18 D. and over in each eye the bilateral operation is always to be preferred — in the hope of recovering the lost binocular vision.

The operation consists, according to Fukala, of a simple discission with the needle, and at first he was in the habit of doing a small iridectomy. Other operators have dealt with the lens in various ways, removal by a linear incision, thorough discission and subsequent removal through a corneal, splitting the lens with a Graefe knife, etc.

If very little or no reaction occurred, or if the lens did not soon become opaque, Fukala again "stirred up" the lens substance as at first. This was repeated as often as needed until the lenticular body was well forward in the anterior chamber. The eye was quieted by atropia and cold applications. As a rule the lens was allowed to undergo gradual absorption, but if increased tension, pain or ciliary injection showed itself, the cornea was punctured with a lance-shaped needle and as much lens matter as possible removed. The best visual results are not to be expected inside of three or four months. Fukala has mostly operated upon young subjects, and has never seen any bad results follow his operations. He has always found that the myopia does not increase and the fundus picture does not change.

Vossius gives a short history of nine cases upon whom he operated. These were chiefly those whom glasses did not help and who were practically unable to do near work of any useful kind. The results were in most cases good. The myopia was decidedly lessened and the visual acuity greatly improved. The author found a remarkable difference in the results as regards the former condition; the refraction decreased from 13 D. in the lowest instance to 28.5 D. in the highest. The case of the latter is worth a brief mention: A student, 20 years of age, has a well marked progressive myopia. There is a strabismus *convergens*, with diminished ocular excursion in all directions. Media clear, albinotic fundi, temporal conus and no macular changes. Under cocain discission of the right anterior capsule followed by considerable swelling of the lens.

Six days afterwards a linear extraction with a Beer's knife. There was a slight anterior synechia, but the corneal wound entirely healed and the eye became quiet. There is a round pupil.

Before the operations O. D. had 30 D. of M., and V. $= \frac{20}{100}$; O. S. had 25 D. of M., and V. $= \frac{20}{60}$; after the extraction on the right side V. R. $= \frac{20}{60}$, and *the refraction is now only — 1.5 D.* The patient can now read small print at the normal distance with-

out a glass, can distinguish people across the street, and declares that his perception of color is greatly improved, and is much pleased with the result.

The effect upon the refraction of the patient's right eye by this operation was certainly remarkable and unusual. The author points out the fact that skiascopy was used, in addition to the subjective tests, in all the cases as a control experiment, and that no mistake might be made in determining the exact refractive condition. He explains the loss of 28.5 D. of M. by the peculiar appearance of the lens; it was almost round like that of the new born infant, and was in all probability moulded into that shape by long-continued spasm of accommodation.

In dealing with the lens in these nine cases of myopia only two were allowed to undergo spontaneous absorption after repeated needlings. In the others, and especially in those instances where there were signs of irritation following the preliminary discission, the lens was removed *en masse* through a peripheral linear incision. It was found necessary to make repeated punctures of the cornea when portions of the lens remained.

Preliminary cutting through the whole mass of the lens has a very different effect in different cases. Its chief drawback is that it tends to bring about proplapse of the vitreous, and to make it necessary to repeatedly puncture the cornea for the purpose of expelling portions of the lens.

Simple linear extraction is, however, usually effective in removing all the lens matter required in these cases of excessive myopia. It is absolutely safe when strict antiseptic and aseptic precautions are taken, and infection of the wound or loss of an eye is a very rare accident.

MYOMA OF THE CHOROID.

Analogous reasoning would lead us to suppose that myoma of the choroid and ciliary body should be comparatively common. Like the uterus, the interior of the eye is well supplied with the material for the growth of muscular tumors, but nevertheless, ocular myomata are very rare. Guaita, who has looked the matter up finds that in addition to his own case only two others have been published. One of these, however, is widely known from its publication in the *Traite complet* of deWecker and Landolt.

The microscopical appearances of the tumor sections are reproduced in ten micro-photographs that appear to be quite as successful as such pictures commonly are, but so far photography has not

reached that point where, for purposes of illustration, it can successfully compete with metal engraving or even with careful photolithography.

The history of the case[4] is as follows: C. A., 20 years of age, mechanic. Father died of pulmonary tuberculosis; mother alive and healthy. One sister healthy, but four brothers died young. Although not very strong he has a sound constitution, and does not present any organic lesion.

Nine months ago he was first conscious of a slight cloud before the right eye, but this symptom was unaccompanied by pain or irritation. During the past two months in looking at an object with his right eye alone he found that above and below the horizon he could distinguish it fairly well, but towards the center there was quite a large horizontal area in which vision seemed more or less confused, as if he were looking through a cloud. During the past fortnight vision in this region has been entirely abolished, the visual defect gradually shading off until perfect sight is obtained above and below. During this time he has never had any pain except one night, about a month before, when he suffered a little and there was considerable lachrymation; in the morning the eye had become quiet again.

The right pupil is markedly dilated so that the iris measures only 2 *mm.* across; it is unaffected by light, but a 1 per cent solution of eserin contracts it. The ocular tension is somewhat augmented, and the deep vessels of the sclera are slightly engaged.

Upon the sclerotic, down and in, and occupying the space between the insertions of the internal and inferior recti muscles 4 *mm.* from the corneal margin is a rounded elevation 3 *mm.* in diameter. At this point this tumor shines through the sclera with a reddish-yellow color.

With the upright image the retina is seen to be detached, but is not markedly pushed forward. When the patient looks down a portion of the retina is seen to be adherent. At this spot also there is seen a yellow-red reflex, the background being dotted over with circumscribed spots of black pigment giving to the elevation an ill-defined outline approaching the globular. This corresponds in position to the elevation visible externally, and reaches to the equator of the globe. Some small retinal vessels ramify over the surface of the tumor.

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[4] Prof. L. Guaita: Mioma della coroide. Clinica oculistica della Universita di Siena. *Annali di Ottalmologia*, Anno XXIV, Fasc. 1, 1895.

The writer, although uncertain as to the diagnosis, suspected either a subretinal cysticercus or a choroidal neoplasm. As he was unable to decide at once he kept the patient under observation, but when, after two weeks' time, and repeated examinations, he was unable to discover any movement in the yellowish-red body, (although he found that it was slowly increasing in size) he fell back upon the diagnosis of tumor of the choroid. This, of course, means sarcoma in the great majority of instances, and the patient having been advised consented to an enucleation which was performed with good results.

The eyeball was thoroughly hardened in Flemming's fluid and after immersion in various graduated mixtures of alcohol was divided into two hemispheres, the section passing through the center of the tumor.

Without entering into a tiresome translation of a very full report given of the microscopical examination it is sufficient to say that the sclera was not involved in the tumor, and that the latter extended from the ora serrata to the equator. It measured 8 *mm.* in its long diameter by five in breadth. Including the choroid it was from $1\frac{1}{2}$ to 2 *mm.* thick. It presented in this way an irregularly elliptic body having its greatest diameter antero-posteriorly. Its external surface was the choroid; its internal the altered retina. The choroid was not especially adherent to the tumor except at certain points.

There can be no doubt but that the mass of the neoplasm is made up of fibro-cellular muscular tissue with its characteristic elongated nuclei. These fibers are mostly longitudinal and unite to form bundles between which may be seen fusiform spaces lined with endothelium which are probably lymph channels.

The tumor contains pigment distributed throughout its mass, but it seems to be less abundant at its external surface where it joins the choroid. The vascular supply of the new growth is rather scant, the vessels found being branches of those normally supplied to the choroid, little, if any, enlarged. The nutrition of the neoplasm seems to have been carried on chiefly through the lymph spaces just described.

The retina mostly presented the changes found in detachment of that coat.

It is the opinion of the author that the mass of the tumor developed from the longitudinal muscular fibers of the choroid. One of the micro-photographs, the most satisfactory of the whole ten, shows very beautifully how these fibro-cellular bands spring

from the choroid, radiate into and form part of the mass of neoplasm. In other parts of the tumor presented to the choroid the new growth appears to be pedunculated upon the surface of the former, an appearance due to its attachment by similar bands of new ingrowing muscular tissue.

THE VOSSIUS METHOD IN CATARACT EXTRACTION.

The writer pins his faith to a method without iridectomy, and gives as a reason for the omission that it is much simpler for the patient and more ideal for the operator. The former suffers less pain and retains his round pupil. The visual result is, however, no better than extraction with iridectomy, while the healing process occupies about the same space of time with both methods. The chief value claimed by Vossius for the simple form of extraction, that he is now in the habit of doing, lies in the position and charactor of his incision. Instead of confining it, as do most operators, to the clear cornea, he makes a conjunctival flap, thus procuring early closing and smooth healing of the wound, combined with quicker consolidation of the operation scar. All these factors are important in preventing infection of the wound, in forestalling prolapse of the iris and its incarceration between the lips of the incision.

The pupil is dilated *ad max.* by atropin-cocain. Then with a Graefe knife he carries his incision through the limbus cornea so as to include about one-third of its circumference and makes, in cutting its way out, a conjunctival flap 3 or 4 *mm.* long. Turning over the mucous flap he freely cuts through the anterior capsule with the cystitome, now, holding a repositor in the left hand he presses the iris well back into the periphery of the anterior chamber, and while it is held in this position proceeds in the usual way, by careful pressure upon the lower border of the cornea with a rubber spoon, to coax out the lens. The iris is now replaced, the conjunctival flap fixed in position and made as smooth as possible. After a slight compression of the globe with the cotton wool, a few drops of eserin solution are dropped into the eye and a bandage applied.

A CASE OF REPAIR OF A LARGE RUPTURE OF THE LENS CAPSULE WITHOUT THE FORMATION OF CATARACT.

Liebrecht[5] gives the following instructive history: A workman came to him with the complaint that he could not see distinctly

[5] Liebrecht: Ein geheilter Fall von isoliertem grossen Linsenkapselriss ohne Kataractbildung. *Beitraege zur Augenheilkunde*, XVIII. Heft, p. 75.

with his left eye since he had been struck (several hours before) on the outer aspect of the globe by a large piece of iron while hammering.

The conjunctiva at the site of the injury is dotted with small hemorrhages, but there is no wound. The cornea is everywhere smooth and shows an unbroken reflex. The anterior chamber is normal, the iris is not injured and the lens is in its proper position. The anterior capsule is ruptured in an oblique direction — downwards and outwards from above inwards. When the pupil is widely dilated the rupture stretches from one edge of the iris to the other. At the center the rupture is quite 1 *mm.* wide, closing in at either end. At the site of the rupture the lens is slightly cloudy; in other situations transparent. After a few days numerous punctate opacities were discovered in various parts of the lens. The lens capsule lies in folds which have formed at right angles to the rupture, but do not extend to the equator. The ophthalmoscope shows the nerve-head and vessels to be distorted, as in irregular astigmatism.

The progress of the case was as follows: the edges of the wound became more swollen and appeared whiter, but on the other hand, the opacities between them gradually disappeared until a week after the accident they could not be seen even after a careful examination with a lens and a bright illumination. The whitish edges of the wound now approached one another from the pointed extremities of the rupture. With a magnifying glass delicate fibers can be seen running from and along its edges, some of them losing themselves in the depths of the lens. The space between the edges of the rupture continued to be filled in by processes from its sides until in four weeks time the lens opacity could no longer be seen through it. This left a broad, white mass of fibers which faintly marks the old site of the rupture. In the course of time this collection decreased considerably in size and lost its white, glistening appearance, but there remained a fold in the capsule above and below the wound.

Seven months afterwards V = $\frac{5}{15}$, for the near, Sn. III, slowly.

It may with certainty be assumed that repair took place through proliferation of the capsular epithelium at the edge of the rupture. This must have occurred early to have defended the lens matte from destructive attacks of the aqueous humor, and the epithelial layer must have been very thin and transparent. The fibrillar processes and the mass of cells were also propagated from the epithelial tissue of the anterior capsule. Liebrecht made a micro-

scopical examination of the lens and capsule in cases of similar injury to rabbits' eyes and found the same marked proliferation of the capsular epithelium

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GLAND-LIKE BODIES BENEATH THE ANTERIOR LENTICULAR CAPSULE.

The writer claims that in the case described in his paper[6] he was able to distinguish glandular structures in the anterior capsule of the lens.

K. B., 60 years of age, had since childhood suffered from repeated attacks of inflammation in his right eye, and for twenty years has had a large gray opacity in the cornea. His vision is very defective in this eye. Ten years before he suffered an injury in the left eye which produces a temporary and partial blindness. A month ago, probably as the result of exposure to cold while profusely perspiring patient became suddenly blind. A dark cloud appeared before his left eye and he had to be led home.

In the left eye the condition was as follows: Palpebral conjunctiva slightly red and swollen. Arcus senilis, but cornea otherwise normal. Anterior chamber deeper below than above. Pupil an irregular oblique oval reacting promptly to light, and widely dilating (round and regular) under the influence of homatropin.

On the anterior surface of the lens are seen small, guttate bodies of a delicate grayish-white color. These are mostly round, but some of them are oval; others kidney-shaped. With a magnifying lens these appear to be spheroidal bodies, perhaps 0.5 *mm.* in diameter, but both their shape, size and color vary with the amount and kind of illumination. The surface of these small swellings as well as of the lens capsule elsewhere, reflects a glimmering almost pearly white color. As a rule the small bodies are distinctly separated from one another by normal lens capsule; in other cases they are so closely packed as almost to coalesce. The largest number is found at the anterior pole of the lens, but there are also many to be found at the margin of the dilated pupil. The lens capsule itself is otherwise smooth and transparent.

With reflected light these bodies show as small dark points, and appear smaller than they do under the oblique illumination.

In both the anterior and posterior parts of the lens are slight striæ to be seen, but these are mostly peripheral. These are opacities in the vitreous and detachment of the vitreous. V. = fingers at 4 *m.*

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[6] A. Sachsalber: Drusen der vorderen Linsenkapsel. *Deutschmann's Beitraege*, 18 Heft., 1895, p. 660.

An iridectomy was done and the patient further treated with steam baths, rest in bed and pilocarpin injections. This seemed to improve the vision somewhat but the lens changes remained the same.

The writer thinks that the small bodies above described are identical with those described by H. Müller and Otto Becker,[7] and which are regarded by the former as very like the papillæ of Descemet's membrane or the isolated glands in the vitreous layer of the choroid. That they occupied the space immediately under, and were covered by the capsule itself, is made certain by the behavior of the light reflected from them and the surrounding structures.

[7] Untersuchungen ueber die Glashäute des Auges, etc. Gesammelte u. hinterlassene Schriften, 1872, I. 254.

REPORTS OF SEVEN INTERESTING EAR CASES.[1]

By D. Milton Greene, M. D.,
OF GRAND RAPIDS, MICH.

EYE, EAR, NOSE AND THROAT SURGEON TO BUTTERWORTH HOSPITAL, U. B. A. HOSPITAL, HOLLAND HOME FOR THE AGED; LECTURER TO ST. MARK'S AND U. B. A. TRAINING SCHOOL FOR NURSES; CHAIRMAN SURGICAL SEC. STATE MED. SOCIETY, ETC.

IN 1892, in a paper read before the Am. Med. Asso., I reported five cases of mastoid suppuration complicating acute suppurative otitis media. Four of the five cases were operated upon and pus found in the cells and antra. All recovered except the one unoperated. In this one an autopsy proved the diagnosis. In none of these cases was there external swelling, redness, edema, or pain over the mastoid, and the diagnosis was made in a manner original with the writer, so far as I know or could learn from anything that had been published up to that time. It had long been my belief that many people had died from meningitis, cerebral abscess, and pyemia, complicating middle-ear disease, whose deaths were attributed to other causes. Both before as well as since that time I have certainly seen death result in several cases from such complications. In the paper referred to I advocated early operation as a safe surgical procedure, and stated that I did not consider the operation, *per se*, dangerous to life, and I am still of the same opinion. Since my paper was published I have received many letters and reprints from prominent otologists indorsing the views expressed therein, and numerous cases are now recorded of a similar nature. Some have even gone farther and advocated opening the mastoid in obstinate cases of chronic suppurative otitis; while

[1] Read before the Michigan State Medical Society.

others advise the operation as a means of diagnosis in acute otitis media, so as to be sure no pus is allowed to remain in the cells undetected. It is not my purpose, however, to discuss the merits or demerits of this operation. Believing that a report of several cases which present points of unusual interest would be the best means of promoting discussion of this important subject, and to get these cases on record is my only excuse for this paper, and if you will bear with me a few moments I will report them in brief.

I wish to say while passing that the statistics bearing upon this subject fully prove the gravity of suppurative middle-ear disease and I will quote briefly from a few of the many. Though a large per cent of cases of acute suppurative otitis media recover under ordinary or even bad treatment, we never know what case may be complicated and prove fatal.

Dr. Clark, of Chicago, reports a case of thrombo-phlebitis of the lateral sinus, jugular and innominate veins with metistatic abscesses in the lungs causing death. There had been no complaint by the patient of ear disease at the time, but an autopsy by Dr. Hektoen revealed mastoid abscess as the cause of the trouble, which condition had not been suspected, but was supposed to have been malarial fever. Any inflammatory process attended with pus formation in such close proximity to the brain, especially in the deep recesses of the skull like the middle ear and the mastoid antrum, should not be regarded lightly, as is too frequently the case. I fear that la grippe is now the pack-horse for bad diagnoses as malaria has been in the past. The doctor is too apt in obscure cases attended with fever to content himself by presuming it to be that unaccountable la grippe.

Dr. B. Alex. Randall's tabulated statistics of 4,785 cases of ear diseases treated in the Philadelphia Polyclinic, of which 3,914 were middle-ear disease, he found

Simple acute	223	
Suppurative acute	457	
Suppurative chronic	918	
Empyema of mastoid	11	76
Caries of mastoid	40	
Caries of tympanum	16	
	2,074	

Which shows in 2,074 cases serious conditions in 76 cases, or 3.5%.

Pitt found in 9,000 consecutive autopsies, 158 deaths due to suppurative otitis.

Bezold found in 325 cases of ear disease 1.5% of deaths.

Barker, from London hospitals, in 8,020 consecutive autopsies found 179 deaths from suppurative otitis.

Pierce found, in the University Hospital, London, in 820 cases of acute and chronic otitis media a mortality of 2.5%.

The statistics of mastoid disease and operations published by Dr. Frank Allport, in the *Journal of the Am. Med. Asso.* of 1892 and 1893, tend strongly to prove that death following mastoid operation is due to the condition for which the operation is performed and is not due to the operation itself.

Case I. Mrs. J. H. M., 42 years of age. Was called in consultation with Dr. J. A. DeVore and got the following history: Had always been quite well, but never strong; had earache and some discharge from the left ear two years before, following an attack of la grippe, but soon recovered; was taken with la grippe again about two months ago and left ear became painful, membrana tympani ruptured and pus has been discharging ever since in considerable quantity; has considerable pain by spells; temperature 104.2; has a sense of fulness in left ear and feels dizzy. On examination I found considerable swelling in the auditory canal; one-half of drumhead had ulcerated away; some tenderness on pressure over the mastoid; there was extreme swelling and edema over mastoid; could not hear the voice even in a loud tone; heard tuning fork over mastoid. As all signs and symptoms were present which are regarded as pathognomonic of pus in the cells, I advised operation, which we did, under chloroform. Made a curved incision close to the insertion of the auricle, from the tip of the mastoid to a point above the ear. Commencing immediately behind and on a level with the external auditory meatus, I separated the periosteum and all soft tissues from the back wall of the auditory canal, clear to the bottom of the tympanic cavity. With a hollow chisel and mallet I then exposed the cells and went into the antrum, which was fully $^3/_4$ inch from the surface, then chiseled through into the tympanum at the bottom. We found cells and antrum full of lymph, but no pus. The next day pus flowed freely from the antrum and cells. She made a rapid recovery, and in a few weeks was coming to my office with a glass drainage tube in the mastoid. In

four months the hole in the drum had healed and hearing was perfect, even better than in the right ear, which was somewhat affected by catarrhal deafness. At no time after the operation did her temperature go to 101 until it was caused by a little abscess at the point of the mastoid; after opening which her temperature remained normal.

This case is of interest as it bears upon a diagnosis of pus in the cells. Pain, swelling, edema, and redness over the mastoid, which have hitherto been our main reliance and guide to a diagnosis of pus in the cells, in this instance utterly failed us, and no pus was found.

Case II. Mr. R. V., 19 years of age, referred to me by Dr. Boot, called with his brother at my office at 1 P. M. Said he had fever a year before—had earache and discharge from the ear for a month or so, when it ceased; had no more trouble until about three weeks ago, when his ear began to discharge; had never suffered pain during the last attack, but had been working every day at wood carving. He said, "this morning when I got up I felt stiff in my jaw, and it was hard for me to open my mouth." On examination, I found temperature 102; pulse 100; pus flowing so fast that it would fill the canal in twenty minutes or less. No granulations obstructed my view, and I could see clearly a swelling from the upper and back of tympanum, which crowded the membrane far forward and downward. There was some swelling of the back wall of the auditory canal, out as far as the beginning of the cartilaginous portion; had no pain or tenderness or swelling over the mastoid, but there was a distinct swelling back of and below the mastoid process, which felt boggy, and was as large as a silver dollar. I sent him to the hospital and had him thoroughly prepared for operation. Under chloroform anesthesia, in presence of several physicians, I incised the soft tissues, as in the case just reported, and with hollow chisel and mallet I opened into a cavity through about $^1/_4$ inch of solid bone, $^3/_8$ of an inch back of and on a level with the meatus auditorus externus. A flood of pus filled the wound and ran over the surface, and was followed by a gush of venous blood. My first impression was that I had opened the lateral sinus, but the hemorrhage soon ceased. With the blunt end of my chisel I probed the cavity carefully, but to my surprise could feel no internal bony wall. I dressed the wound with bichlorid gauze. Saw him next morning, temperature 100; pulse 100; felt "well." Carefully irrigated the cavity and ear; there was very little pus. In the

evening I examined the cavity in the mastoid through the opening in the bone (which was $^1/_2$ inch in diameter) by the aid of a head mirror — there were no mastoid cells — and the inner bony plate was entirely absorbed away, leaving the duramater bare over a surface $^7/_8$ by 1 inch or more in diameter. The lateral sinus to the extent of an inch or more could be plainly seen, and at a point about midway of its exposed portion a small vein could be seen where it had been broken off, and about $^1/_{16}$ of an inch remained attached to the lateral sinus. Drs. Boot, Fuller, Kelly, and others, examined the case and saw the condition described above. His temperature never went to 100 but once after the operation, and in six weeks he left the hospital well, but wearing the drainage. He made a perfect recovery and works at his trade. I see him frequently, and his hearing in the operated ear, which had been *nil* for a year before the operation, is now nearly normal, a year after the operation.

Case III. Mrs. J., had earache, profuse discharge following poultices applied over the ear. Saw her with the family physician. Temperature fluctuating between 99 and 102 or 103; had some pain in mastoid region, but no swelling, drumhead freely opened about one-third, having sloughed away; swelling from upper and back wall of tympanum, pressing mallius far forward and downward; profuse flow of offensive pus. Advised opening mastoid, which they declined. I therefore abandoned the case. She was treated by the family physician by irrigation, politzeration, etc., for about six weeks, when the discharge nearly ceased. Temperature had been from 99 to 101 during this time. At this time she had a chill, and fever followed; temperature dropped to normal, and in a day or two became sub-normal. She could retain nothing in her stomach, vomiting whenever she moved, was dizzy and stupid. I was called again, found her tongue red and dry, pupils contracted. Told the doctor she had evidently meningitis, with effusion or cerebral abscess caused by suppuration in the mastoid, and that I believed she would die in a very few days. She soon sank into coma and died in two or three days. In this case no autopsy was secured. I firmly believe that an operation at the time I first saw her would have saved her life.

Case IV. Mr. Wm. T., 43 years of age, referred to me by Dr. Eugene Boise, had chronic discharge from the left ear; was taken with chills, and temperature ranged from 99 to 103. Was treated for fever for two weeks, when he was turned over to me on account of a profuse and offensive aural discharge and the conclusion by his

physician that his trouble was due to that cause. I found him somewhat delirous, restless and a profuse offensive discharge of pus mixed with blood issuing from his left ear. Diagnosed mastoid disease from signs and symptoms similar to those in case No. 3; advised operation as the only chance of saving life, which was, perhaps, at that time one in a thousand. They were strangers in the city, having recently come from Chicago, and wanted counsel. Two aurists and two other physicians saw him with me. One aurist thought he would recover without operation other than making a free opening into the tympanum, which he did with a Graefe knife, the other aurist and the two physicians thought there was no hurry about operating, and we left the case in care of one of the physicians. He grew constantly worse, continued delirious, vomited, became comatose and died in five days. No autopsy was had to settle the diagnosis, but he presented unmistakable signs and symptoms of abscess of the brain with pressure; had spasm in right arm and right side of face.

Case V. S. B. E., 17 years of age, referred to me by Dr. Graves; had been at Ottawa Beach for a month late in the season; had been bathing every day and diving frequently; came home with slight purulent discharge from both ears, and had some pain, but not severe; both drumheads were perforated; there was considerable swelling in the external auditory canal. I treated him a few days, when the discharge ceased, but swelling increased and extended over one mastoid. I concluded he had external mastoid periostitis and sent him to the hospital. Temperature 102. I made Wild's incision, $1^1/_2$ inches long through the periosteum, and pain ceased; temperature dropped to 99. Two days later the other side became painful and swollen and his temperature raised to 101. Under chloroform I made Wild's incision, and pain abated at once and temperature fell to normal, and in a week he left the hospital and made an uninterrupted recovery. This was a case of external mastoid periostitis *pure and simple*, and was caused by myringitis and extension of inflammation along the periosteum over the mastoid. This, like case No. 1, goes to prove that pain, swelling, edema and redness over the mastoid does not necessarily indicate that there is pus in the cells or mastoid antrum.

Case VI is of special interest for several reasons, which will appear later.

February 21st was called by Dr. Rutherford to see Miss H., 24 years of age. She had been suffering with pain in both ears for

twenty-four hours. On examination I found both drum membranes inflamed and bulging from accumulation of bloody serum in the tympanum. Temperature 101; pulse 100. No cause could be assigned for the ear trouble. The left ear being the worst I punctured the membrana tympani and a bloody serum flowed freely. Was called again in a few hours and opened the other drumhead with like effect as in the left ear. There was extreme congestion in both ears, and though the pain was relieved for the time, it soon returned in both ears. I ordered leeches and morphin to relieve the pain. Next day temperature 98.8 to 99. The next four days the temperature was below 99. Pain continued intermittent and pus was flowing from both ears. Fifth day temperature rose to 101. Complained of deep-seated pain in both ears and extreme sense of pressure. Leeches had been applied. Ordered blisters over the mastoids which filled well. Sixth day had counsel of several physicians. All symptoms aggravated. I determined to open the mastoid which I did on the left side, in the usual way, and found cells and antrum full of lymph and blood and some small deposits of pus. I opened from the antrum into the middle ear through the bony wall. I then made a Wild's incision on the right side. Pain was relieved on the left side, but continued on the right. At the operation we noticed just in front of the tragus a red spot as large as a 25-cent piece surrounding a recent leech bite, but thought it nothing serious. At night, to my surprise, it had spread to the size of three inches in diameter extending over the cheek and eye. We saw we had a case of erysipelas as a complication. It spread over face, head, neck, and in a few days had enveloped shoulders and had extended down the back as far as the last dorsal vertebra, but had not attacked the wounds over the mastoids which were made within a portion of skin, which had been blistered by cantharides. As the patient was delirious and seemed to be growing worse, and erysipelatous process steadily going on, not being checked by carbolic acid, sub-cutaneous injections of boric acid, etc., I determined to circumscribe it by cantharidal collodion. This was suggested by the fact that, though the whole head was enveloped by erysipelas, the part which had been blistered escaped. This treatment cut short the process, and the inflammation never went beyond the blister which extended in a line over the breast and back encircling the whole body. In a few days the erysipelas was entirely cured. At the end of thirty days she still had severe pain in her neck and arm and some in right ear. Pulse 100; temperature 101.4. The symptoms caused by erysipelas had made it impossible for me

to judge what part of her fever, delirium and pain were due to the ear or mastoid, and what part to the erysipelas, but as her temperature was above 101 and erysipelas cured, with pus flowing from her right ear, with swelling at the upper back part of tympanum bulging forward and downward, I decided to open the mastoid; which I did and evacuated two or three drachms of pus, and pain was relieved. From this time she went on to complete recovery. I removed a small sequestrum of necrotic bone from the wound in left mastoid during this time. She was completely deaf in both ears before the operation, but the ear first operated was soon restored to hearing, while the hearing in the other ear is still defective.

If I had such a case to treat again I should open the mastoid on both sides at the time of the first operation.

Case VII. Mr. M. D., 50 years of age, Hollander. When a boy had earache and a discharge from his left ear for several years which finally healed and quit discharging. Had no more trouble until he was 30 years of age, when it began to discharge pus in a small quantity. Had been deaf in the left ear for twenty years or more. About six months before had considerable pain, and a swelling took place just above and in front of the helix. He consulted an aurist who ordered poultices applied. This treatment was continued for some weeks. The doctor then punctured the swelling with Graefe's knife and evacuated some pus. He repeated this several times and continued the poultices. Patient, after some months, fell into the hands of another aurist who made an incision over the mastoid about half an inch long but got no pus. The case did not improve and the second aurist turned the case over to me, saying if I could do anything for him to do it. At St. Marks hospital I made a careful examination, and by probing through the incision first made by the Graefe knife I detected a hole in the temporal bone. Temperature 102; pulse 100. I then prepared him for operation, and under chloroform I made an incision about an inch in length in the abscess just in front of the helix, and by passing my finger in could feel a hole in the temporal bone about $^3/_4$ of an inch in diameter, and the duramater exposed; bone was roughened and I diagnosed caries of the temporal bone. I then extended the incision upward and forward over the temporal to the frontal bone commencing at the lower end of the first incision close to the helix; I made an incision about 3 inches in length upward and backward to the masto-occipital junction. I then made an incision from the latter down behind the ear to the tip of the mastoid. Keeping close to the bone and raising the periosteum I laid back the two upper

flaps, and in like manner separated the attachment of the ear and carried it forward and downward on the jaw and neck. Now having temporal bone exposed I separated it from the parietal and frontal bones, and with Volkman's spoon, gouges and chisel I removed the whole temporal bone while Dr. S. C. Graves held up the temporal lobe of the brain with his hand. The only portion of the temporal bone not removed was a small portion of the inner end of the petrous portion traversed by the internal branch of the caroted artery. The piece was loose and movable and with my thumb and finger I could grasp it and move it about. I should have removed this little piece but for the advice of some of the physicians present who thought the man would not get off the table alive, though he appeared in good condition. I said that the whole bone should be removed to be successful, for it is a well-known fact that as long as any of a temporal bone remains which has been once attacked with caries, the process will continue and it will cause suppuration and abscess. Under the press of opposition I left the piece of petrous bone. I then brought the flaps together after thorough washing with bichlorid 1.3000, I put in a large rubber drainage and united the flaps by interrupted sutures. The temperature fell to 99 and did not go above 100. The wound was kept well irrigated and in two weeks it had healed and pushed the drainage tube out. Four days after the operation the patient sat up, and in a week was walking around the ward. Temperature and pulse normal, and appetite good. About a week after the wound was healed completely he had some deep pain in the region of the tragus. Temperature began to fluctuate. A small swelling occurred over the junction of malar portion of zygomatic process with the temporal. I made an incision and removed a small piece of temporal zygoma and the wound healed. Pain and temperature continued for four days when the temperature fell to 97, and in two days he died. At the autopsy which was made by Dr. H. Lupinski we found between the duramater and pericranium in the temporal region on the operated side a fibrous deposit about as thick as the skull which had been removed, which formed good support and protection to the brain. The arachnoid membrane was free in the temporal region except in five points immediately beneath the hole first found in the temporal bone which was evidently caused by the punctures made with the Graefe knife, or a small silver probe. Surrounding the piece of petrous bone which was left there was an abscess full of rather thin pus from which septic absorption and septic meningitis had taken place, which caused death. The only signs of meningitis were at the base of the brain in the region of the petrous bone which was left.

This case is of special interest in connection with the consideration of mastoid and middle ear disease for the following reasons:

1. It was caused by suppurative otitis media.
2. It illustrates how nature may protect the brain and membranes, and tolerate pus in contact with the duramater.
3. It shows the great tolerance of the temporal region to operations.
4. It is fair to suppose that had the little portion of petrous bone been removed at the time of operation he might have recovered. Before the operation the patient could not open his mouth or move the lower jaw; a week after the operation he could open his mouth and masticate his food.

I hope my paper will not lead you to think that I advocate the too free use of the chisel and mallet, for I certainly do not, but as many cases are obscure and liable to be overlooked I would advise vigilance and care, so that dangerous cases may not pass unnoticed until they have done irreparable damage.

Of the whole number of cases of ear diseases which come under my care a very small per cent require mastoid operation, although I believe I do the operation more frequently than most aurists. I believe I treat an unusually large number of ear cases and am called especially in those cases where operation seems necessary. The cases above reported are a few of the many operations I have done within the period of time covering those cases and they have been selected for variety and to illustrate the point which I have hitherto made that external swelling, edema, redness and pain over the mastoid are not necessarily signs and symptoms of pus in the cells, neither is it safe to depend upon them as a guide to a diagnosis of pus in the cells or to the necessity of an operation. The topography of the temporal bone varies to such a degree in different skulls that it is not always easy and in fact never possible to locate accurately the lateral sinus, semicircular canals and other important anatomical structures, which fact makes it necessary for an operator to be very careful in his manipulations and to have acquired a faculty of judging each case by itself. Skulls vary in form and size, but not more than do the mastoid cells and antra. A comparative study of

many hundreds of skulls, and experience in operating in the ear and upon the mastoid, have brought me to the conclusion that in skillful and experienced hands the operation of opening the mastoid is free from shock and comparatively free from danger, if done under antiseptic precautions. The operation should never be done with a drill. The chisel has not the elements of danger that the drill has. With the chisel and mallet one can work carefully and see what he is doing, with the drill the operation is done in the dark, so to speak The antrum should always be reached and opened freely, and if there is pus in the cells they should be carefully scraped out unless found quite tough and sound, as is frequently the case when the operation is done early. The earlier the operation is done the less extensive it has to be. In all my cases never but one died after operation, and this young lady had been entirely unconscious for forty-eight hours, and was nearly moribund before the operation was commenced.

SYSTEMATIC ACOUSTIC GYMNASTICS IN THE TREATMENT OF DEAFMUTISM AND CASES OF NERVE DEAFNESS IN GENERAL; A SYSTEM AS ADVOCATED BY PROFESSOR URBANTSCHITSCH, OF VIENNA.*

By J. Steele Barnes, M. D.,
of Milwaukee.

I DESIRE to call attention to a method that promises better results than any plan of treatment that has heretofore been instituted for improving the condition of a class of otological cases that have hitherto received but little benefit from treatment. In fact, the deaf ears have received the least attention, but now effort is being directed to a development of hearing in the deaf ears. I shall neither go into the etiology or pathology of deafmutism or of this method in its relation to deafmutism. I simply present this brief paper to again call attention to the possible results to be obtained by acoustic gymnastics, and to outline the manner of conducting the treatment.

The method is no longer one of speculation and experiment nor can it be looked upon as the fad of an enthusiast, as the results obtained by patience and perseverence clearly demonstrate it to be of sound and practical value.

The idea of aural gymnastics in the treatment of deafmutism did not originate with Professor Urbantschitsch, but so far as we know, with Bewus[1] over a century and a half ago. Later, Itard[2] in 1821, and Toynbee[3] in 1860, reported success with

* Read at the Forty-Ninth Annual Meeting Wisconsin State Medical Society, West Superior, June 19-21, 1895.

[1] Beck. *Ohrenheilkunde*, 1827, p. 73.

[2] Itard. *Traite des Mal-ad. de l'oreille*, 1821, Paris, T. 2, p. 476.

[3] Toynbee. *Die Krankh. des Gehörorgh.*

cases treated by similar methods; but to Urbantschitsch we are indebted for developing the idea and proving its great value. His attention was called to what was possible of accomplishment with the method by his remarkable success in the case of a deaf-mute boy. He says,[4] "Several years ago I obtained a surprising improvement of hearing by systematic acoustic gymnastics in the case of a deaf-mute boy referred to me for treatment. At first he was able to hear only vowels and single syllables spoken loudly in his ear; later, after a year, he could understand complete sentences spoken in a moderately loud voice and finally was able to receive ordinary, not deaf-mute instruction."

There is nothing extraordinary or intricate in the method. It is virtually a vocal training of the auditory nerve whereby a gradual irritability of the nerve for sound waves is set up. It is, as it were, a bringing into life and activity a hitherto lifeless nerve.

At the Vienna Policlinic Professor Urbantschitsch devotes an hour daily to the training of deaf-mutes by acoustic gymnastics, and it was there my good fortune to receive personal instruction and witness the surprising results obtained. It was indeed interesting to note the gradually increasing perception of sound in ears that were at first apparently totally deaf.

Instruction begins with the vowels. Two are selected, usually *A* and *O* as they are the most readily understood. The one to be used is first indicated to the patient by the lips and then in a loud and prolonged voice is spoken directly into the ear. The patient in the beginning may not hear a sound, but persistent and continued effort will, even sometimes at the first sitting, enable him to arrive at a differentiation, of two sounds. While it may not be a complete differentiation, the sound of *A* conveys a different impression to the mind than the sound of *E* shown by the effort of the patient to repeat. Again, this result may not be obtained until after several sittings. As soon as the differentiation of the vowels is mastered consonants are added to them, gradually increasing to long syllables, then to words and finally complete sentences.

As progress is made the distance from the ear at which the

[4] Urbantschitsch. *Wiener Klin. Wochenschr.*, 1893, No. 29.

exercise is conducted may also be gradually increased, and after a few sittings it is best to dispense with the lip method making the exercise purely acoustic.

While perception of sound is essentially necessary to gaining any progress in a case, conception of sound makes the perception easier, exerting a marked influence upon the rapidity of improvement. One may have perception long before he has any conception of a sound, but the sooner a sound conveys intelligence to the brain the quicker and more readily is that sound always distinguished. Therefore from the very beginning the attempt should be made to convey to the patient a meaning with every sound, to have him realize that every sound has an intelligent impression for the mind. In connection with the conception of sound I noticed a corresponding improvement in phonation and articulation. It was much easier for a deaf-mute to reproduce a sound that conveyed some intelligence to his mind than one that did not. I will quote from Dr. Goldstein[5] who says: "A case is cited of a deaf-mute girl, who, during one of these acoustic exercises, was given the word *Anna*, the name of one of her sisters. This word was plainly heard and correctly repeated, yet when the meaning of the word was asked, the patient was unable to associate the idea of her sister's name with the word heard. When the explanation was given the young girl's surprise was great, and from that time a rapid improvement was noted as the patient made every effort to combine the spoken word with the idea implied."

Regarding the frequency and duration of the sittings I believe Urbantschitsch has found that short, continued practicing of from five to ten minutes, gives the best results. Practice should be daily at first, gradually increasing in frequency rather than lengthening the duration. However, each case must be a study of itself.

The pitch and intensity of the voice suited to the case must be determined and great care taken that the irritability of the nerve be not exhausted, as some ears are exceedingly sensitive and tire very easily. Should this occur, all practice must be stopped for a few days, otherwise more harm than good

[5] Goldstein. *Archives of Otology*, Vol. 24, No. 1, 1895.

will be done. To give you a more definite idea of this method I could quote you the record of several cases in which primarily a vowel spoken loudly directly into the ear could not be heard; but at the end of six to eight months' practice could understand and repeat correctly entire sentences, the instructor being five feet distant, and giving no aid by the lip method. However, I will not consume time in describing cases already published.

The results to be obtained depend largely upon the systematic manner in which the exercise is conducted, but the condition of the hearing and the personal factors of the case to be practiced naturally exercise great influence also. In the first place there must be an existing ability to hear and an ability for development of the hearing. According to Urbantschitsch[6] however, "complete deafness is seldom found among deaf-mutes, and even if at times the ability of hearing seem entirely wanting, if only a trace of hearing is aroused, a further development is possible. The existing trace of hearing can be increased to the perception of a tone, the perception of a tone to a vowel, and this again to the perception of a word." It has been repeatedly demonstrated that in deaf-mutes who appeared totally deaf, there has resulted from systematic acoustic gymnastics a gradual improvement up to a high degree of hearing.

This method is applicable also to cases of acquired deafness resulting from scarlet fever, typhoid, cerebro-spinal meningitis, diphtheria, measles, etc., and not alone for the nerve, but for the long standing obstructive sequelæ do I believe it to be of utility. I have at present under treatment a man 44 years of age, who came to consult me about his right ear. Said his left was totally deaf and that he had not heard with it for the last twenty years. Examination showed beginning sclerosis, right middle ear; in the left ankylosis of ossicles with probable impaction of stapes; high tone tuning forks heard on mastoid; could distinguish vowels shouted directly into the ear; history dates from an attack of scarlatina. Treatment was instituted for the right ear and daily practice with acoustic gymnastics for the left, with the result that

[6] Urbantschitsch. *Wiener Klin. Wochenschr.*, 1894, No. 19.

after seven weeks he is able to hear words distinctly at a distance of three meters, complete sentences at one meter. I have also used vibratory massage, but attribute very little to that as the case was of such long standing, and the improvement so marked and rapid the first two weeks. Later I hope to report this case more in detail.

The possibility then of improvement is certain; the limit of that improvement uncertain. As yet it is impossible to say to what extent the improvement in hearing may be carried. Only time, patience, and perseverence will demonstrate.

307 Grand Avenue.

TRANSLATIONS FROM FOREIGN CURRENT OTOLOGICAL LITERATURE. (ABRIDGED).

By H. A. Alderton, M. D.,
OF BROOKLYN.

TRI-CHLORACETIC ACID IN CHRONIC SUPPURATION OF THE EAR.

Dr. W. N. Okuneff, St. Petersburg, (*Monat. f. Ohrenh.*, Jan., 1895). Tri-chloracetic acid is superior to others, *e. g.*, chromic acid, in that it does not spread, and after its use syringing with salt solution is unnecessary. The crystalline form of the acid makes its use very difficult, but following Cholewa, I use a common iron wire upon the end of which a loop is formed. A small crystal is engaged in the loop and passed once or twice over the glass of a burning lamp; it requires only a slight warming until the crystal melts in the loop as in a frame. The acid spreads quickly at the point of application, and it is better to remove the super-abundant acid by syringing. The cauterization is very painful so that a 10% or even a 15% cocain solution should be used previously to anesthetize the parts. The previously warmed cocain solution should be left for two to three minutes in contact with the diseased parts, during which time, two or three syringes should be prepared with luke-warm water, and the crystal engaged in the loop of the applicator. Then, with the aid of a hard rubber speculum, apply the crystal, after drying the ear thoroughly, to the mucous membrane of the middle ear as well as to the edges of the perforation. The mucous membrane and the edges of the perforation become white. Then to prevent the spreading and to remove the super-abundant acid, quickly syringe. The patient must meanwhile

cleanse the ear at home. After drying ear, I blow aristol, boric acid, etc., therein. Generally this procedure should be performed one to two times per week. But in those cases where one wishes to promote the growing together of the perforation, cauterization should not be done oftener than every eight or nine days, as too frequent cauterization destroys the new growth of granulation and connective tissue. Between applications syringings with disinfectants are made or in moderate otorrheas, drops (sol. resorcin 2%) applied, or the ear is left alone.

I have used this method mainly in cases of chronic suppuration with large perforations in the *M t*. In most, treatment by other remedies had failed. Others presented granulations upon the *M t* or the adjacent walls of the canal. Finally in cases where one wished to stimulate the epidermal edges of the perforations, in order to lead to its cicatrization.

In all cases treated as above, the otorrhea with its usual odor disappeared. The pus loses quickly its unhealthy color. The granular irregularity disappears, the mucous membrane becoming even and smooth, the small polyps also disappear. But with moderate sized polyps the cauterization fails, and here it is necessary to operate. Dry perforations, with epidermal edges, can in a shorter time be brought to cicatrization than by the use of other remedies and more permanently, if the middle ear process is at an end. It is not only necessary to restore the *M t* by cicatrization but also to restore its qualities of integrity and invulnerability in order to protect the inner parts from external unfavorable influences. Tri-chloracetic acid has the capability of doing this. Also after cicatrization the hearing may often, by systematic treatment, be brought on the road to a normal condition. This is especially true in childhood.

I believe that in persons from 5 to 25 years of age, by this means, always the end of the otorrhea, the cicatrization of the perforations, and the return of the function of hearing can be brought about.

In forty-two cases under my personal observation treated by this method, in thirty-eight the otorrhea ceased 90.4%, in two marked lessening 5%, in two slight lessening 5%; in twenty-three cicatrization of the defects and perforations, *i. e.*, in more than half of the cases. As in most of the cases the cauterizations were not systematically conducted, but were more or less occasional and irregular, so can one suppose that the results would be still more pronounced were the cauterizations systematically performed.

Where the physician does not hesitate to bestow a half hour upon his patient, and is not afraid of tediously progressive manipulations, then I repeat, can one always succeed in obtaining the complete cure of the chronic otorrhea and the re-establishment of the hearing.

Very true is this in childhood where we can always count upon the regenerative capability of the membrana tympani.

COMMUNICATION UPON DEEP BRAIN ABSCESS CONSEQUENT UPON ACUTE DISEASE OF THE TEMPORAL BONE.

Dr. H. Eulenstein, Frankfort a. M., (*Monat. f. Ohrenheilk.*) By searching the literature the author found eighteen cases in all, of otic brain abscesses occurring as sequelæ to acute diseases of the petrous bone. Also adds to these one observed by himself, operated upon with favorable results.

Dr. Eulenstein has only brought those cases together which without doubt are to be reckoned as acute, and which originated in direct connection with acute diseases of the petrous bone, or in which the origin was taken from an already disappeared acute ear disease, the traces of which were still clearly perceptible.

The eighteen cases are described by the following authors: Moos, Farwick, Guerder, Bezold, Schmidt, Gruber, Braun, Picqué, Baginsky-Gluck, Jansen, Ferrier-Horsley, Hoffmann, Grubert, Polo, Truckenbrodt, Joel.

The etiology of the acute temporal disease was measles in two cases, angina one, typhus one, influenza three, instillation of aqua fortis one, foreign body one, no cause given in nine.

The location of the abscess corresponded in all of the cases to the side of the diseased temporal bone, and was six times on the right side, eleven times on the left, and in one the side was not given. This fact, the more frequent occurrence on the left side is remarkable, especially as Koerner in his excellent work upon otic brain diseases, found in brain abscesses in general a remarkable preponderance upon the right side. The number of cases collected here is too small as contrasted to the great number of otic abscesses in general, to conclude therefrom a contrary opinion in regard to brain abscesses contracted from acute diseases of the ear.

Thirteen abscesses were situated in the temporo-sphenoidal lobe, five in the right side and eight in the left; four in the cerebellum, one in the right, three in the left; the situation was not given in one case. We observe here, as prevails in the total of otic brain diseases, that the preponderating majority had their seat in the temporo-sphenoidal lobe.

Multiple abscesses were observed three times, and of these, two abscesses in one case were observed in the cerebellum; in two cases two abscesses in the temporo-sphenoidal lobe. From the reports of these cases, there existed no ground for the belief that pyemia had existed, the abscesses also exhibited metastases.

The appearance of multiple otic brain abscesses in general is of great rarity, as Koerner has shown (in sixty-two cases of cerebral abscesses occurring only five times; in thirty-two cerebellar only four times), so is the relative frequency (three in eighteen cases) in the hitherto observed acute cases astonishing.

The reports give no valuable information as to the size of the abscesses or the existence or absence of a pyogenic membrane.

Of the so-called general symptoms of brain abscesses, expressive of the gravity of the disease, the state of the body temperature is of importance. Fever of irregular altitude occurred in thirteen cases, three cases ran without fever, and in two cases the temperature chart was not given.

Headache, one of the most constant symptoms of brain abscesses, from increased intra-cranial pressure, was explicitly noted as absent in one case.

Retardation of pulse was noted three times, irregularity once.

Choked disc occurred three times.

In most of the cases the sensorium was more or less benumbed.

The focus-symptom, so valuable for the diagnosis, existed eight times in the thirteen temporo-sphenoidal abscesses. There were observed interference with speech (in the left-sided temporo-sphenoidal abscesses); paralyses, as paralysis of the opposite extremities and of the facial, hemiopsia, ptosis, paralysis of the abducens of the same side. Moreover, photophobia, incontinence of urine, increased tendon-reflex, conjugated deviation to the right (in right-sided temporo-sphenoidal abscess), hemianesthesia, and hyperesthesia were observed.

Besides the general symptoms (fever, headache, benumed sensorium) in the cerebellar abscesses, unilateral paralysis of the opposite facial was observed in one case, torticollis in one, supression of breathing in one.

Death resulted in twelve cases, (eight temporo-sphenoidal, three cerebellar, one not noted); cured by operation six, (five temporo-sphenoidal, one cerebellar). A fatal termination occurred earliest in seven days after the beginning of the ear disease, latest about six months after beginning.

In the six successfully operated upon cases, recovery followed in from eight weeks to six months after the beginning of the primary disease.

As complications were observed, thrombo-phlebitis of the lateral sinus alone twice, thrombo-phlebitis and lepto-meningitis together once, pachy-meningitis externa three times. Of these, two with pachy-meningitis externa were cured by operation, one with sinus-phlebitis died after operation. Without operation both the other cases of sinus-phlebitis died, also lepto-meningitis as well as one case with pachy-meningitis.

The sinus-phlebitis was situated on the left two times, in one case the location was not given. In the three cases with sinus-phlebitis only one abscess was present.

The mastoid process was affected twelve times, as follows: periostitis, acute caries, empyema, etc.; in six cases there existed no mastoid disease.

In the thirteen temporo-sphenoidal cases mastoid disease occurred eight times, the latter being healthy five times; in the four cerebellar cases mastoid disease occurred three times, being healthy in one; (the situation of the abscess was not given in one case).

Of the eighteen cases, fourteen in all were operated upon as follows: mastoid operation alone three times, mastoid and opening of brain abscess nine times, opening of brain abscess alone two times.

Recovery occurred in both the cases in which the brain abscess alone was opened, the mastoid process being healthy. Of the nine cases in which both mastoid and brain were operated upon four were cured and five died. The three cases in which the mastoid alone was operated upon, as well as four cases not operated upon, all died.

The results of the operations, so far as concerns the brain abscess, was as follows: Eleven brain abscesses (eight temporo-sphenoidal, three cerebellar) were operated upon, five died (two cerebellar, three temporo-sphenoidal); and six were cured (one cerebellar, five temporo-sphenoidal). In eight cerebral abscesses operated upon five were cured, of three cerebellar one recovered.

The cause of death in the five fatal operative cases was once œdema of the brain, once rupture into the lateral ventricle and meningitis, hemorrhage and softening of the cerebellum once, no autopsy was made in two.

As regards brain pulsation, so important diagnostically, in the operative cases is mentioned once explicitly as existing, five times as being absent, while five times nothing relative to this condition was noted. From experience with brain abscess in general it may be said that absence of brain pulsation indicates the presence of pus, whilst its existence does not prove the opposite.

As regards the frequency of brain abscesses sequel to acute ear diseases as compared to those sequel to chronic suppuration, a collection by Grunert shows that otic brain abscess occurs in 9% through acute ear disease; in the Berlin clinic Jansen has observed only one brain abscess in 2,650 acute suppurations of the ear.

Dr. E.'s case shows the following points of interest: It proves percussion of the mastoid process to be an excellent diagnostic help; that in the absence of suppuration from the ear, by the intactness of the coverings of the mastoid process, only by the etiology, the location of the pain in connection with the result of percussion could be determined the diagnosis of disease of the mastoid. Also the necessity for early operation. Also in acute diseases the *usually valuable indications indicating opening of the mastoid process do not suffice.*

Also that at no time in Dr. E.'s case was the almost constant condition of drowsiness or unconsciousness present. The complete absence of fever during the attack is of further interest. So far as Dr. E. knows, the singultus which persisted for eight days, is an as yet unobserved early symptom, for the existence of which he can offer no explanation. The lowering of the body temperature upon the opposite half of the body to the abscess was also of interest. Further, the lowering of the hearing of the opposite side is a rarely observed symptom, although one should more often expect it since the center for hearing for the opposite ear lies in the temporo-sphenoidal lobe.

In the after treatment, cautiousness is required in the use of the tampon as it may, though loose, restore the pressure exerted by the evacuated pus; still one is constrained to resort to it whenever a somewhat severe hemorrhage occurs.

OBSERVATIONS ON HEREDITARY SYPHILIS OF THE MIDDLE EAR.

Chambellan, (*Journ. de Clin. et de Ther. Infantiles, No. 26*), demonstrates that hereditary syphilis does not affect the internal ear only, but may affect also the middle ear, and that without the internal ear being involved.

In the case of a child, 12 years of age, who had been treated in different clinics for otitis media sclerosa, the treatment, politerizations, had led to no amelioration. It was then, March 23, 1893, that they brought the child to me, then $12^1/_2$ years of age, under treatment since about $4^1/_2$ years of age. She spoke nasally like a deaf person. Both tympanums were manifestly sclerotic. No trace of suppuration and there was no history of such. Watch heard in O. S. on contact, in A. D. at 4 *cm.* Tinitus in both ears. Heard better in dry than in damp weather. The integrity of the internal ear was absolute.

Under treatment the condition continuing to grow worse, the patient was questioned about her parents and it was found that the father died at 28 years of age, probably of cerebral syphilis, from that to infer that the young girl was affected by the taint of hereditary syphilis was only a step. The signs of Hutchinson were entirely absent. In spite of all, Chambellan prescribed 2 grams of iodid of potassium daily and mercurial inunctions.

Under the influence of the specified treatment, the condition of the patient ameliorated progressively; at the end of three months the mother said the child did not seem to be deaf at all on dry days.

The author concludes:—

1. Observation shows the existence of hereditary syphilis in the middle ear, a fact not noted until now. In the case, the child was $7^1/_2$ years of age at the beginning of the disease.

2. This hereditary syphilis of the middle ear takes the form of otitis sclerosa.

3. This otitis sclerosa of heredo-syphilitic origin has a slow course, and may, if not entirely cured, at least retrocede in a satisfactory way as against labyrinthic otitis of the same origin, which has a rapid course and leads almost always to complete deafness. The first is therefore less grave than the second.

TWO CASES OF HEMATOMA OF THE EXTERNAL AUDITORY CANAL IN CHILDREN.

A. Barbier, (*Journ. de Clin. et de Ther. Infantiles, No. 23*). Hematoma of the canal is less known than of the auricle.

The author has observed two cases, one in a boy 12 years of age, the other in an infant 10 months of age. On examination, one could see at the entrance of the auditory meatus a tumefaction, a little hard, but yielding lightly to pressure, obstructing all the orifice and retaining the habitual coloration of the skin of the region. With both patients these sessile tumors were inserted on the inferior wall of the canal. The development had been slow and progressive; entirely painless. These symptoms suffice for the differential diagnosis.

In the two cases the author practiced ablation of the tumors with the bistoury; the cure was rapid.

The tumor removed, one saw that the fundus of the canal and the tympanum presented their normal aspect. Barbier, from the etiological point of view, notes that the two children were of feeble constitution, and manifestly tainted with scrofula.

A RARE CASE OF VERBAL DEAFNESS.

G. Masini, (*Bollet. d. Mal. dell 'Or., etc., No. 6, 1894*). A young man, 18 years of age, of feeble constitution, never had any important disease; subject since two years to losing, from time to time, the faculty of comprehending what is said to him, though he has retained the normal perception of sound. During these attacks he hears, but no more understands than if the language were foreign; he can repeat the words pronounced in his presence, but cannot seize their meaning; he can understand by signs, can even speak, but his ear does not control his tongue and his language is broken and disconnected. These phenomena last several minutes, then suddenly disappear; and as suddenly he comprehends anew what is said to him. These crises are accompanied neither by *aura* nor vertigo. The patient seems to remain conscious of what passes around him.

These disorders are evidently of cortical origin. Are they a manifestation of hysteria or do they represent a form *larvée* of epilepsy? Masini does not pronounce clearly, but seems to lean more toward the first hypothesis, in favor of which one could invoke the existence in this young man of a concentric contraction of the visual field.

A METHOD TO RENDER MICROSCOPIC PREPARATIONS OF THE HEARING ORGANS TRANSPARENT.

Dr. L. Katz, (*Berliner Klin. Wochens.*), has for many years used a method in the preparation of the hearing organs, that exhibits the membranous labyrinth quite isolated by means of the old spirits preparation, in which the connective tissue of the membranous labyrinth is very well preserved by the alcohol. Afterwards the preparation is decalsified by a 15% muriatic acid solution, and after two or three days a quantity of nitric acid (about 15 to 100 *Cc*) is added to this muriatic acid solution, the surrounding bone becomes macerated in such fashion that when placed in water, the membranous labyrinth can easily be extracted from it with the aid of the needle.

By the method Katz has lately used it is possible to bring the tympanic contents and also the membranous labyrinth clearly into view.

A temporal bone is placed about four to six weeks in chromic and osmic acid (0.25 chromic acid, 0.5 glacial acetic acid, 0.25 osmic acid to 100. aqua).

By this the nerves, after some time, become stained blue-green and intensified, and so can be much better observed. Then the hardened preparation is washed out and placed in about a 20% solution of nitric acid for about fourteen days when it is decalsified. It is then washed again and placed first in a 90% alcohol solution and afterwards in absolute alcohol.

All superabundant tissue, especially the dural covering of the bones, must be removed, because they obscure the transparency.

After the preparations have lain in absolute alcohol for three or four days they are put in xylol for twenty-four hours, this latter having the property of making the bones completely transparent.

The sections made should not be of greater thickness than 1 *cm.*, because of microscopical reasons, but may be of any length or width.

Katz has recently modified the method in such way that a quite distinct osmic staining of the nerves within the membranous labyrinth is attained. The stapes is first extracted in such a fresh petrous pyramid and then the pyramid put in osmic acid, the fluid presses through the oval window with ease into the vestibule and into the turns of the cochlea, and distinctly stains all the nerve elements. When in pathological cases it is desired to preserve the stapes, then through the superior semi-circular canal the fluid is injected by means of a Pravaz syringe. It is essential that the

osmic acid is applied primarily to the fresh specimen. The osmic staining is so distinct that such a preparation satisfies almost ideal claims, especially for study purposes.

In order to submit the preparation to the most favorable possible investigation, it is placed in glass cells, with parallel walls. These cells are protected from the air by moderately strong cover-glasses. The cells are filled with Canada balsam, the preparation laid in and the cover-glass closed with avoidance of bubbles.

After one or two days the cover-glass through the balsam becomes adherent to the cell, and remains so unless brought near warmth.

One gains surprising views by means of the so-made preparation when brought under proper microscopical enlargements (four or five times).

ABSTRACTS FROM CURRENT FOREIGN OTOLOGICAL LITERATURE.

By T. Melville Hardie, B. A., M. D.
of Chicago.

Szenes contributes to the Sixty-sixth Congress of German Naturalists and Physicians (*Monats. f. Ohrenheilk*, October, 1894, *Jour. of Laryng.*, January, 1895) his experience in the treatment of 130 cases of ear disease with glycerin of carbolic acid and menthol. Instillations of 10% to 20% carbol glycerin were used in the initial stage of acute otitis media. If used warm before perforation takes place they frequently cause a retrogression of the process. A weaker solution should be used if perforation has taken place—10% and 15% solutions of menthol in oil are used in furuncle of the meatus and also in diffuse otitis externa. It frequently prevents recurrences. It should always be used after incision and removal of the contents of the furuncle. It may best be introduced on an absorbent cotton tampon and may be left in the meatus for twenty-four hours.

Gradenigo (*Ibid*) believes that many cases of sclerosis develop as a consequence of hereditary syphilis and that these differ only in greater malignity from the ordinary form met with in females with a tendency to hereditary ear disease or hereditary tuberculosis. The diagnosis may sometimes be difficult on account of the absence of any other symptom of hereditary syphilis, but we must always be on the lookout for it in cases of progressive deafness which comes on rapidly, with involvement of the inner ear, in young persons in whose families there is no tendency to hereditary tuberculosis or deafness. While this may be the only manifestation of the syphilitic diathesis it may be accompanied, as in the typical form, by malformation of the teeth and interstitial keratitis. In very rare cases it accompanies gummata and ulcerations of the nose and pharynx.

EXPERIMENTS ON THE IRRIGATION OF THE TYMPANIC CAVITY.

Bing (*Jour. of Laryng.*, April, 1895) carried out experimental irrigation with colored fluids on the temporal bones of adult bodies in which the cartilagino-membranous portions of the eustachian tube with the pharyngeal orifice had been carefully preserved. The membrana tympani was freely exposed and in its lower half a small hole was made with a pin, or else a wide paracentesis cut was made, or a portion of the membrane was excised. The preparation was then held as far as possible in the natural position. The colored fluid was injected through a catheter placed in the tube, and its outflow into the meatus was observed. Then the tegmen tympani and the cortical covering of the mastoid was chiselled away, and the tympanic and mastoid cavities so laid open were examined. It was found that with a wide paracentesis cut or a large opening in the drum most of the fluid ran out, and no coloration was found either in the upper cavities of the tympanum or in the antrum, while with a small perforation opening in the drum less fluid came out into the meatus, but, on the other hand, it could be found in abundance in the antrum and the mastoid cells. He concludes from this, with certain reservations:—

1. In the case of large openings in the drum, irrigation of the tympanum cannot quite achieve the intended object.

2. There is ample confirmation for the assertion that the outflow of injected fluid is sufficiently free, and easily effected through the opening in the drumhead.

On the treatment of purulent inflammation of the middle ear in which there is a small perforation at the point of a mammillaform projection on the drum. He recently treated cases of this kind which are known to run a complicated or at least protracted course by means of a drop of liquor fer. sesquichlor. applied to the seat of the perforation by means of a probe. Rapid recovery followed. The medicament caused no particular reaction; it exercised a powerful astringent effect; the otorrhea ceased in a few days; the apertures quickly cicatrized, and under antiphlogistic and absorbent treatment perfect restoration took place.

Gomperz said that brilliant results often followed irrigation of the tympanic cavity through the tube in acute and chronic suppurative inflammations, but that the result could not be counted upon; that under certain circumstances it could do harm and lead to actual danger, and he would, therefore, advise that irrigation through the tube should be quite given up. In earlier years he

saw cases of acute inflammation of the middle ear, in which, when the irrigations through the tube were commenced, ostitis of the mastoid process supervened in spite of a free outflow, and improvement took place only when this treatment was given up.

Gomperz made large openings in the drumhead of the dead body and injected a weak solution of ferro cyanid of potash through the tube with the usual pressure, so that the fluid could run out through the meatus. He was then able to make out, by means of the reaction with perchlorid of iron, that the fluid had reached the narrowest passages of the diploe nearest the cranial cavity. From this he thought it proved that by irrigation the infective organisms could be driven deep into the osseous parts into which they otherwise would not have reached. Then he dwelt upon the possibility that in cases of defect of the tegmen tympani or antrum the fluid might reach directly under the dura mater.

Reinhard recommended irrigation from the tube only in chronic cases, not in acute suppuration of the middle ear, whether the acute cases were of the muco-gelatinous or the purulent kind, especially when complicated with caries of the median and inferior wall of the tympanum. One condition must always be present: absolutely free egress through a sufficiently large opening in the membrane, which should be carefully ascertained beforehand by means of the air douche.

Politzer thought that Gomperz's fears were exaggerated. Anatomical experiments are not always applicable to pathological conditions as purulent inflammatious matter was always present in the mastoid antrum, and hence no fluid could be driven in. To give up these injections would be a great loss to therapeutics, as only by these means in a number of obstinate cases could recovery be brought about. The case of cerebral abscess in which death had suddenly taken place after injection was more easily explained by the shock induced by the catheterism and injection.

Scheibe agreed with Gomperz and Reinhard as to the danger of the tympanic injection through the tube in acute middle ear suppuration, but considered it necessary in the chronic inflammations.

IRRIGATION OF TYMPANUM.

Brieger considered the copious tympanic injections through the tube beneficial only when all inflammatory manifestations should have subsided. In contrast to the treatment recommended by Bing for the papillary perforations he advised active dilatation of the

orifice, and mentioned that in two cases he had removed the prominence with the snare for the purpose of histological investigation, and had thereby brought about a very good result.

Gruber thought that in chronic exudative inflammations the use of the injections should not be given up. There were cases in which the exudation could be removed from the drums in no other way, and moreover, the irrigations serve to allay the severe pain. In acute cases he did not use them for fear of driving the exudation into the mastoid cells. In chronic cases the danger is less, as in the majority of cases, there is already inflammation in the mastoid cells.

Szenes had seen two good results from the use of injection in acute inflammation of the middle ear, though he thought they should not be used as a rule.

Pins had good results from the use of irrigation in chronic and subacute cases excepting in one case where cerebral complications and probably abscess of the brain occurred. Vertigo and death followed one day after the second irrigation. The method is of great use if the perforation of the drum is sufficiently large to allow free exit for the fluid.

Gomperz replied to Politzer that when in pathological cases the cell-spaces of the mastoid process are filled with pus, the latter was exposed to a higher pressure during injection, which was a matter of some importance.

TREATMENT OF THE ATTIC IN CHRONIC SUPPURATION.

Gomperz, on the results of the conservative method of treatment in chronic suppuration of the upper cavities of the tympanum.

Gomperz reminded his hearers of his statistics (*Monatsschrift*, 1892 and 1893) of the results of the non-operative treatment of forty-nine cases in his private practice, which he, as far as it was possible, had followed up, and of twenty new cases. Of these latter, nineteen have so far recovered after an average treatment of twenty days that all secretion and formation of granulations have disappeared; that the mucous membrane up to the interior of the attic looked pale, soft, even epidermized, and the function of the ear had been raised very materially. He considered the excision of the malleus and incus as a very important extension of our therapeutic resources, but only applicable in suppurations of the tympanic attic when recovery cannot be brought about by the

conservative method, and that the recurrence of cholesteatomatous formations cannot be guaranteed against by excision of the malleus and incus. Why he believed so firmly in the conservative method is that so many subjects of cholesteatoma formation present constitutional dyscrasics, such as anemia, scrofula, apical catarrh, lupus and hereditary lues.

EXTRINSIC AURICULAR REFLEXES.

Bonnier read, at a meeting of the Paris Society of Laryngology and Otology, February, 1894, (Journal of Laryngology, March, 1895) an elaborate paper on auricular reflexes; of these the intrinsic reflexes are largely of theoretical interest only; the extrinsic, of more general value, are here reproduced.

1. *Auricular nerve.* From the close association of this nerve with the phrenic is derived the explanation of the hiccough, clucking or eructation sometimes occasioned by irritation of posterior wall of the external meatus.

2. *Pneumogastric branch.* Irritation of this branch causes the auricular cough.

3. *Auriculo temporal.* Through this nerve diplopia, and even optic atrophy, have been traced to disease of the ear. In a case of herpes of the eye and of the tympanum the central affection was probably located in the Gasserian ganglion.

4. *Glosso-pharyngeal.* Irritation through this may produce nausea and vomiting.

5. *Chorda-tympani.* Implication of this nerve may cause repeated swallowing of saliva, or burning or tickling feelings on the corresponding side of the tongue.

6. *Utricular nerve.* This nerve enables the animal to judge of the position of the head, the direct sensorial source of equilibration, and communicates with the cerebellum (vermis superior) from whence pass by the spinal bundles of the middle peduncle, the fibers which communicate centrifugal impulses to the fundamental fasciculus of the antero-lateral columns. There are numerous commissural connections. Most of those fibers of the utricular nerve which do not go to the cerebellum terminate in the internal or vestibular nucleus, the bulbar center for subjective direct cephalic orientation and the starting point of all the reflexes associated with vertigo. From this nucleus fibers run to the superior olive, which in its turn, gives off an important bundle to the nucleus of

the external oculo-motor (sixth nerve). Hence the oculo-motor reflexes observed in ear disease, with or without vertigo, such as strabismus, diplopia, pupillary contraction, nystagmus and disturbances of accommodation. Inversely violent excitation of the retina has been known to produce deafness.

7. *Saccular nerve.* Modifications of pressure are perceived by means of this nerve. It may, therefore, influence the respiratory and cardiac rhythm. Through it may be brought about the palpitations, feelings of oppression, epigastric pains, vomiting, and epileptiform or hysteriform attacks which accompany labyrinthius commotion.

8. *Cochlear nerve.* Epileptic attacks have been known to be excited by musical sounds, and startings are similarly produced by sudden noises.

It will thus be seen how many reflex disturbances may arise from affections of the ear. Local treatment of a simple kind is often all that is required in order to allay them.

Gomperz (*Monatsschrift für Ohrenheilk. October, 1894*) *on the recognition of protrusion of the bulb of the jugular vein into the tympanum during life.*

Up to the present, five cases of dangerous hemorrhage following paracentesis of the membrane from perforation of the bulb of the jugular vein projecting into the tympanum have been published by Gruber, Ludewig, Hildebrandt, Seligmann and A. Brieger, the last one ending fatally. For the preven tion of this casualty the writer recommends careful inspection of the membrane before paracentesis, and he sets forth the symptoms of this anomaly in cases in which the membrane was not inflamed, founding his views upon observations made in this direction. Blue discolorations in the lower half of the drum membrane, which, from their posterior extent and color, have nothing in common with the shadow of the niche of the fenestra rotunda are not uncommon. We find the blue spot either circular or bi-convex always close to the lower periphery of the drum, the convexity extending upwards and forwards. Finally he recommended for consideration whether in view of the danger of penetrating the bulb in an inflamed or opaque membrane, paracentesis should be practiced in the artero-inferior quadrant in a direction from behind forwards.

Zaufal thought that in cases of such hemorrhage we ought not to be content with simply plugging as deep as the membrane.

Small tampons ought to be pushed through the opening as far as the injured spot.

Politzer had seen hemorrhage into the drum after paracentesis of the membrane from perforation of the veins accompanying the nerve of Jacobson.

Habermann had seen dehiscence of the floor of the tympanum with projection of the jugular bulb reaching as high as the horizontal semi-circular canal.

LINING MASTOID CAVITY AFTER CHISELLING.

Reinhard experimented thus: After the opening of the middle ear by two parallel horizontal incisions in the posterior cuticular wall a flap was formed whose base was situated at the external auditory meatus. He continued these incisions outwards in the concha so far that thereby the external meatus was enlarged, the skin wounds were closed by means of sutures, and the flaps from the posterior wall of the meatus were fixed against the posterior wall of the osseous cavity from the meatus by means of tampons the tongue of the skin was pushed backwards into the large mastoid cavity so as to paper it.

He had made use of the above method in two cases of circumscribed caries of the attic and of the antrum without cholesteatoma. The patients had to wear the bandages only a short time and were soon able to leave the hospital—within ten to fourteen days. The after-treatment is easier because of the increased width of the external meatus, and from the cosmetic point of view this seems to him to give the best result after the chiselling operation. Those cases in which a persistent retro-auricular opening is to be striven for are excluded (cholesteatoma of the mastoid, extensive tubercle, caries, etc.) The method is suitable only for circumscribed caries of the ossicles and of the attic and antrum, in which the opening of the cavities of the middle ear is necessary. In properly selected cases he considers this the ideal method.

ABSTRACTS FROM CURRENT AMERICAN AND ENGLISH OTOLOGICAL LITERATURE.

By Leonard A. Dessar, M. D.,
of New York.

FURUNCULOSIS OF THE EXTERNAL AUDITORY CANAL.

Dr. S. MacCuen Smith, (*Medical News*, January 19, 1895), in a practical article on this subject, expresses the opinion that aural furuncle is both infectious and contagious, with a tendency to recurrence from auto-inoculation, and that it occasionally manifests itself epidemically. He briefly summarizes the treatment as follows:

1. As antiphlogistic measures, use the leech or blister in front of the tragus, and hot antiseptic irrigation when indicated. Avoid the use of poultices.

2. As local applications, cleanse the canal with alcohol and insert an ample tampon of cotton-wool saturated with camphor or naphthol, renewing this every twenty-four hours, or oftener if required. This is at once antiseptic and analgesic.

3. As constitutional remedies, give tonics and alteratives with the especial recommendation of arsenic in the form of Fowler's solution. This should be administered in increasing doses until its physiological action is obtained.

4. As an operative procedure, make a free incision through the boil, and divide the periostium down to the bone. This will prove necessary in well-advanced and chronic cases, especially when pus has already formed.

CLONIC SPASM OF THE TENSOR TYMPANI.

Dr. R. C. Heflebower (*New York Medical Journal*, March 16, 1895) reports a case of this rare and obscure affection which he is inclined to regard as a distinct form of chorea minor. The patient, a married woman, 35 years of age, had suffered from a chronic

otorrhoea of the right ear of many years' standing, with a large perforation. After four weeks' treatment the discharge was arrested, and the perforation much diminished in size. In the left ear the membrane was a little lax, the reflex absent; hearing distance for watch sixteen inches. For several years the patient had complained of a clicking noise in the ears, accompanied by a twitching of the muscles of the throat. This had become gradually worse and annoyed her greatly, being exaggerated by any marked exertion. On inspection of the right membrane no movement could be observed, although the clicking noise could be faintly heard. In the left ear, however, a very perceptible indrawing of the central and lower part of the membrane could be seen at the time of each click; the muscles of the soft palate contracted synchronously with the tensor tympani. The larynx was not in the least implicated. In each ear the clicking was simultaneous with that in the other, and was never rythmical. It came on at irregular intervals, and was louder at certain times than at others.

The author had occasion some time ago to observe a second and more marked case of this affection, the sound being perceptible at a distance of two feet at times. The patient, a man of mature age, was considerably affected mentally, bordering upon insanity, and this of interest in connection with the fact that the father of the first patient was a confirmed lunatic.

A FEW OTOLOGICAL DON'TS.

Dr. N. S. Roberts, of New York, (*Medical Record*, June 1, 1895) makes the following practical remarks on the treatment of aural affections: Don't treat earache with chloroform, laudanum, or camphorated oil, they are inefficient and may excite local inflammation; it is better to fill the ear with hot water, with or without glycerin, apply a poultice, and, if pain persists, apply a leech to the tragus. Don't advise or permit a patient with profuse otorrhœa to constantly wear cotton in the ear, it causes retention of the discharge, and operates against that diligent attention to cleansing and other treatment which would otherwise be given. Don't use the galvano-cautery in the auditory canal, it is liable to be followed by such consequences as necrosis, ulceration, otitis externa, or stenosis. Don't blow insoluble powders into the ear when there is a purulent discharge through a small perforation, it may appear to stop the discharge, but it does so usually by occluding the perforation, and may be followed by worse conditions.

Don't neglect to look for ear complications in all eruptive fevers, typhoid fever, diphtheria, and low types of pneumonia. Don't politzerize through nares in which there is stored up foul catarrhal mucus. Don't politzerize with much force in sensitive patients, or those having a thin, translucent drum membrane. Don't neglect to keep watch of the mastoid prominence in all cases of purulent otitis, and if tenderness, heat and swelling are found, to take measures to subdue a probable incipient mastoiditis. Don't overlook, in chronic ear disease, constitutional conditions such as lithiasis, scorbutus, tuberculosis, and syphilis. This precaution applies likewise to diseases of the nose and throat.

THE ELECTRO-PNEUMATIC MASSEUR IN TINNITUS AURIUM.

Dr. Chevalier Jackson, of Pittsburg, (*Jour. Am. Med. Ass'n*, June 29, 1895) reports the results obtained with the aural masseur devised by himself in the Manhattan Eye and Ear Hospital in the service of Dr. Clemens. Among twenty-two cases of otitis media catarrhalis chronica, associated with tinnitus aurium, treated with this instrument, two cases were unfavorably influenced, and twenty more or less benefited. The relief afforded in some instances was striking, and in one case the noise disappeared after three applications and have failed to return. On the ground of his experience Dr. Clemens regards the masseur contra-indicated in cases where the existing retraction of the membrana tympani and malleus causes an increased intra-labyrinthine pressure.

SUCCESSFUL MECHANICAL TREATMENT OF SOME UNUSUAL AURAL CONDITIONS.

Dr. William B. Marple, of New York, (*New York Medical Journal*, June 1, 1895) presents in detail the history of a most interesting case of aural disease successfully treated by a simple mechanical contrivance. The patient, a young married woman, had been greatly troubled for a year with vertigo which was referred by her in its beginning to the right ear. The vertigo only came on when the head was held in a certain position, *viz.:* tipped to the right, and was so severe that she often suffered from nausea. Always during the persistence of the vertigo her right ear felt stuffy, and the tinnitus which also existed, was worse. The hearing in the right ear for the watch was at first only one foot, while in the left it was fifteen feet. Inflation failed to improve the hearing in her right ear, the membrane of which was somewhat

retracted and thickened. Suspecting that the trouble was due to some tension anomaly, the author made use of a simple device employed by Dr. Clarence Blake, of Boston, in a similar case. He inserted a small piece of rubber dam used by dentists into the auditory canal in such a way that the ends lay against the anterior and posterior wall of the meatus, while the central bent portion rested against the short process of the malleus. Thus a pressure from without was exercised (estimated by Blake to be equal to about fifteen milligrammes) which was in a direction favoring the most complete opposition of the malleo-incudal articulating surfaces. This pressure from without counterfeited in a measure the effect of the traction from within. After the introduction of the rubber spring in Marple's case, examination several weeks later showed that the hearing distance for the watch had increased on that side to twelve feet. The vertigo gradually diminished so that the patient was able to sleep on her right side. At the end of three weeks when the rubber was removed, as a control experiment, the vertigo and nausea returned, but disappeared when the rubber was reintroduced, and this experience was verified on several occasions. After about five months the rubber was replaced by a paper disc inserted against the membrana tympani in the posterior quadrant. This effectively relieved the vertigo, which always recurred, however, when the disc became dislodged. It was permanently removed about one year after commencement of treatment, and since then there has been no return of the vertigo, although the tinnitus has persisted. In two other cases of disagreeable buzzing sounds in the ears when certain musical sounds were struck this symptom has been relieved by the introduction of the rubber spring.

EVACUATION OF THE TYMPANUM.

Donald B. Fraser, M. B., M. R. C. S., Eng., of London, Ont., (*Medical News*, April 27, and May 18, 1895) recommends the following simple method of emptying the tympanum in cases of disease of the middle ear, especially abscess: All that is necessary is a bulb syringe made of good rubber, having a suitable nozzle, and no valves. Compress the bulb by squeezing it lightly, introduce the nozzle into one nostril, press the other tightly against it, direct the patient to keep his mouth closed, release the bulb and allow its expansion to exhaust the tubes and ear cavity, and then press the bulb slightly and get the patient at the same time to blow

against the lips until the cheeks are fully inflated. Thus, inflation and evacuation is performed alternately, the two processes being effected by one instrument.

In the *Medical News*, May 18, Dr. B. A. Randall points out that evacuation of the tympanum can be more effectively secured by the Toynbee method which consists in deglutition with the mouth and nose closed. He cautions, however, against all these methods, which, in his opinion, are rarely of practical value.

DIFFUSE EXTERNAL OTITIS.

Dr. M. D. Lederman (*New York Medical Journal*, May 18, 1895) records a case of severe diffuse external otitis due to syringing the ears with a strong solution of carbolic acid which the patient had been advised to use by a friend for the relief of itching probably due to an aural eczema. When seen two days later there was serous infiltration of the face, marked congestion of the conjunctiva, and diffuse swelling of the auricles, this being especially pronounced on the left side which had been chiefly subjected to this heroic treatment. Serum filled the fossæ of the auricles, and exuded from a false membrane which covered the tragi, anti-tragi, and scaphoid fossæ. Testing with the tuning fork permitted the exclusion of internal ear disease. The watch could not be heard with either ear, but patient distinguished the voice when spoken to in loud tones. Under the author's treatment, consisting of administration of codein for the relief of pain, and the external use of Labarraque's solution as a deodorizer, rapid improvement occurred; later Politzerization was practiced. No narrowing of the canal resulted, and no permanent disturbance of the auditory perception. That absorption of carbolic acid had taken place was demonstrated by the ocular symptoms (muscae volitantes, affection of vision) and the passage of cloudy urine.

ABSTACTS FROM CURRENT NEUROLOGICAL LITERATURE.

By Wendell Reber, M. D.,
OF POTTSVILLE, PA.

OCULIST AND AURIST TO THE CHILDREN'S HOME—LATE JUNIOR RESIDENT PHYSICIAN STATE HOSPITAL FOR INSANE, NORRISTOWN, PA.—FORMERLY CLINICAL ASSISTANT WILLS EYE HOSPITAL; ALSO TO THE EYE DEPARTMENTS OF THE PRESBYTERIAN AND ST. AGNES HOSPITALS, PHILADELPHIA, PA.

THE ALLEGED REFLEX CAUSES OF NERVOUS DISEASES.

Dr. Phillip Coombs Knapp, of Boston, read a paper, bearing the above title, before the meeting of the Philadelphia Neurologic Society, January 28, 1895, during the discussion of which, Dr. William Osler, of Baltimore, said: "With most of Dr. Knapp's conclusions I am entirely in accord. There are several conditions which we cannot, with our present knowledge, explain except on a theory of reflex influences. I refer more particularly to some of the curious phenomena connected with the adenoid vegetations in the pharynx and disease of the turbinated bones, such as persistent asthmatic attacks in children, which are cured radically and permanently by local treatment; also similar spasmodic affections of the bronchi in adults.

"Then we have the fact of a few well-authenticated cures of exophthalmic goiter by treatment of the turbinated bones.

"Of course I think that a great deal of twaddle has been written with reference to the influence of eye strain in producing epilepsy and chorea. Among the cases of chorea submitted to Dr. Stevens and the cases reported by Dr. Ranney there is scarcely a case of genuine Sydenham's chorea. They are cases of habit spasm. That such cases are cured by the removal of nasal and pharyngeal trouble is undoubted.

"On the question of headaches, Dr. Knapp takes, I believe, an extreme view. I think a larger percentage of cases of headache are cured by removal of ocular defects than he is willing to allow."

Dr. G. E. de Schweinitz: "As one of my distinguished confreres has reviewed certain writings of mine with reference to eye strain with this comment, 'it may suit the general practitioner, but it is not in accord with the experience of ophthalmologists,' or words to that effect, I presume I am safe in saying that I should not be accused of too great partisanship on the side of ophthalmic medicine. I am in entire accord with Dr. Knapp in the belief that Sydenham's chorea is not caused by hypermetropia and hypermetropic astigmatism, or indeed by any ocular anomaly, refractive or muscular. As I have elsewhere written, 'the evidence seems quite as lacking that hypermetropic refraction is the basal cause of chorea as it is that the chorea is the cause of hypermetropia.' In a constitution predisposed to chorea I presume eye strain is an important factor in fastening and provoking attacks. Pseudo-choreas are often the result of anomalies of refraction. That true epilepsy is ever produced by refractive anomalies I doubt, although I am quite sure that convulsive seizures of various types, and no doubt some of those which belong to epilepsy proper, are modified and sometimes checked for long seasons of time by the use of proper glasses, prismatic or sphero-cylindrical. The great difficulty resides in the fact that the reports of these cases have been rushed into print before the proper time has elapsed to test the permanency of the effect. The evident conclusion of the matter is that while we may not believe the extravagant assertions that have been promulgated with reference to the effect of eye strain, we do know that in the management of functional nervous disorders it is one of the influences that must be subdued before the treatment of the case is successful, for precisely the same reason that the defective functions of any other organ should be put into proper order in the management of these cases.

"I am not in accord with Dr. Knapp in his estimate, as I understand it, of the value of connecting refractive errors in the treatment of headache. In the first place, I do not believe that the cause of headache can ever be inferred from the position of the pain. The patient may experience the painful sensations from eye strain in any portion of the head — frontal, parietal, occipital or vertical region. Again, the pain is often situated between the shoulder blades, far down in the neck, and sometimes over the precordium. It is a notorious fact that in many cases of the most

pronounced 'eye strain cases,' the eyes themselves and their immediate vicinity have been entirely free from pain. I do not believe that there is the slightest doubt, other things being equal, that fully sixty per cent of functional headaches will be materially benefited or cured by the proper correction of refractive anomalies. * * * I am quite sure we all agree, and I think Dr. Knapp has said, that in the treatment of all of the cases the examination of the eye is of very great importance, precisely as is the examination of the nose, or, indeed, of any other organ, in order to make sure that the investigation of the case has been a perfect one."

Dr. Knapp, in closing: "There is, perhaps, not so much disagreement between my position and that of Dr. de Schweinitz as he thinks. I think I said that I make it a routine practice to refer almost every case of headache to the oculist, and I rather pride myself that in some instances, I have held firmly against the opinion of the oculist that the headache was due to eye trouble, and the subsequent history has shown the correctness of my view. I have gradually been coming to the belief that one of the distinctive symptoms in refractive headaches is the aggravation of the pain by the use of the eyes for near work. Another symptom that seemed to me to be important, although not so conclusive, is the location of the pain. With the routine practice of referring every case to the oculist, I have obtained satisfactory results in a great many cases, but there is still a considerable remainder of cases where treatment of the eyes had not cured the headache. (*Journal Nerv. and Ment. Dis.*, April, 1895.)

PECULIAR IRIS-REACTION IN THE PRESENCE OF POST-NEURITIC OPTIC ATROPHY.

Dr. Geo. M. Gould, (*Journal Nerv. and Ment. Dis.*, April, 1895), propounds a puzzling problem in neurology, as follows: Patient, a girl, 12 years of age, who had been afflicted with obscure spinal and cerebral disease presented, when she was first seen a year and a half ago, intense optic neuritis in both eyes. A year later there was absolute optic atrophy of the typical white variety. The most brilliant and concentrated light thrown suddenly or continuously through the pupil failed utterly to elicit iris-reaction or perception response. But, by seating the child before an open window, the street in front being illuminated by sunlight or diffuse daylight, within half a minute or more the pupils were found to

be of normal size. The parents had noticed that when the child played out of doors the pupils were of the size usual in other people. The motion of the contraction was too slow to observe, that is, one could not positively say that the myotic movement was taking place by watching the pupil attentively. In the same way the widening when the patient's face was turned away from the window and directed toward a moderately lighted room was perhaps more rapid, but still too slow to detect its progression.

Dr. Gould observes: "The problem, a double one, is this:

"*a*. How can the optic nerve be the apparent intermediate of pupillary response when following a peripheral neuritis that produces total blindness?

"*b*. Why does the pupil react to the stimulus of continuous diffuse daylight, and not to that of the most brilliant artificial light."

Without advancing any hypothesis, Dr. Gould then puts the following alternative queries that occur:

"Is it possible:

"1. That there is a localized molecular action of daylight upon the muscular iris-fibers distinct from central neural connection and control?

"2. That the stimulus, generalized, strong and continuous, of the daylight is powerful enough to carry some nervous impulse through the atrophied nerve fibers, and so far as the pupillary centers, but that this impulse is too weak to reach the visual centers?

"3. Had there been originally a synchronous atropic lesion of the optic centers or of the conducting paths beyond the pupillary centers (which would explain the blindness), but that still left a few fibers intact between the retina and corpora quadrigemina?

"4. Is there some hitherto unproved neural connection between the iris, *per se*, and the quadrigeminal bodies?

"5. Is the neural connection (as yet unproved) by means of the fifth nerve?

"6. Is the visual center stimulated via fibers direct from the retina and not by fibers from the pupillary center; in other words, are there distinct fibers that proceed to the pupillary center and end there, the neural impulse not proceeding hence to the visual center, whilst other distinct fibers proceed directly from the retina to the occipital lobe without calling at the pupillary centers?"

[The phenomenon is so unusual that we wish Dr. Gould had put forward some hypothesis for the condition.—W. R.]

NOTE OF A CASE OF SOFTENING OF THE RIGHT ARGULAR GYRUS, WITH LEFT-SIDED PTOSIS.

C. A. Herter, M. D., New York (*Journal Nerv. and Ment. Dis.*, January, 1895), records a case of left-sided ptosis that came to autopsy, when there was revealed an area of softening in the angular gyrus of the opposite hemisphere. History as follows: A man, 60 years of age, admitted to hospital in a state of stupor. Under observation two and one-half days, during which time the temperature ranged from 100° to 102°. There were some signs of tuberculosis of the lungs; there was a bad cystitis; the heart was very weak and rapid, and the radial pulse could not be felt most of the time that the patient remained under observation; right arm and leg quite flascid, and were only slightly moved on painful stimulation; face not involved; both knee jerks lost; no rigidity anywhere; on the left side there was ptosis not complete; the left pupil was slightly dilated and reached less well to light than the right pupil. The autopsy showed the existence of extensive pulmonary tuberculosis with cavities, and pyclitis with suppurative nephritis. In the right hemisphere, just below the interparietal fissure, was a circular patch of softening 1 inch in diameter, occupying the angular convolution. The softening involved the cortex, and to a slight extent the white substance beneath it. Dr. Herter concludes "it is safe to refer the left-sided ptosis to the lesion in the right hemisphere."

[It would seem an open question whether the left-sided ptosis were due to the lesion in the right hemisphere or whether, perhaps, it was not due to a tubercular or other invasion of the nucleus of the left third nerve or some of its filaments in the fourth ventricle. —W. R.]

A CASE OF ALEXIA (WORD BLINDNESS) WITH RIGHT-SIDED HOMONYMOUS HEMIANOPSIA.

L. Burns, M. D., (*Neurolog. Centralblatt*, 1894, Parts 1 and 2), recites an interesting case history. Patient, a woman, 32 years of age. Disease began two years before death with continuous headache; afterwards vomiting, then visual disturbances and vertigo. Five months before death diagnosis of cerebral tumor made. At that time, choked disc in both eyes, typical right-sided hemiopia, associated with short attacks of complete blindness. The patient further presented the symptoms of Wernicke's sub-cortical alexia, or Fruend's "optic aphasia." Comprehension of speech and musical understanding unimpaired. Objects shown are recognized

but can seldom be named, although the first letter of the object's name is not infrequently mentioned. Printed and written letters are recognized, but cannot be named even when the hand of the patient while writing it is guided by the hand of the author. No words except very short ones can be recognized or read aloud (verbal alexia). Autopsy showed the existence of three glio-sarcomata of the left occipito temporal lobe, which, at the time when the described symptoms of alexia were observed, had not yet reached the cortex; at least a craniotomy performed one month before the patient's death and exposure of the whole convexity of the occipital lobe and a large part of the adjoining parietal and temporal gyri did not reveal any tumor. The "choking" of the discs disappeared after the craniotomy, which, the author says, is a further proof of the theory that choked disc is not a true optic neuritis, but is the direct consequence of increased intra-cranial pressure.

OPTIC NEURITIS AS A SIGN OF BRAIN TUMOR.

Wm. H. Wilder, M. D., (*Chicago Medical Recorder*, May, 1895), makes some very interesting observations on the relative value of optic neuritis in brain tumor. He details a study of 161 cases in which either an operation or an autopsy had been performed. The nature and pathology of optic neuritides are reviewed, the theories of Graefe, Schmidt and Manz, Hughlings-Jackson, Leber, Edmunds and Lawford are stated and the points in which these different hypothesis are lacking are set forth.

Dr. Wilder remarks the infrequency of one-sided choked disc. In some cases the neuritis was more pronounced on one side than on the other, and this, in a large majority of the cases, on the side corresponding to the new growth. He is inclined to believe that the cause of optic neuritis must be sought in the irritation of the nervous elements by the products of tissue change in the growth, causing a descending inflammation, or that they cause a direct irritation of the nerve through the medium of the fluids of the optic sheath. He lays stress on periodical attacks of blindness as a clue to possible intra-cranial growth.

Of the 161 cases investigated, 90 were growths of the type of glioma and sarcoma with their mixed forms. Optic neuritis was found in 74.3% of the cases examined in reference to this sign. Out of the 104 cases with choked disc, 24 showed involvement of the cerebellum, whilst in 25 the motor convulsions were the seat of the neoplasm; 90% of the cerebrellar tumors were accompanied by optic neuritis.

A CASE OF TABES, ASSOCIATED WITH REMOTELY PREVIOUS HEMIPLEGIA, AND EXHIBITING UNILATERAL REFLEX IRIS PARALYSIS.

The following case was presented by Dr. Joseph Collins before the March meeting of the New York Neurologic Society, and was fully discussed by the members:

J. J. D., 38 years of age, male, married, occupation policeman. Family history good. Denies syphilis and alcoholism, and there are no traces of either to be found. Has never used tobacco or indulged in excesses. The one pertinent fact in the history is that when about 23 years of age, he suffered an attack of hemiplegia, which disappeared entirely under mercurials and iodids. As Dr. Collins says, "this condition of things, occurring in an individual 23 years old, who was temperate, who had been singularly free from acute disease including rheumatism, who gave no history of injury or exposure or illness, and who made a complete recovery under specific medication, would point to syphilis as the origin."

Dr. Ward A. Holden, of New York, to whom the case was referred for careful examination of the ocular conditions, reports, among numerous anomalies, the following peculiar reaction: "The right pupil of medium size and reacts sluggishly to light, consensually and to accommodation. The skin reflex wanting in both. The left pupil very small and does not respond to light directly or consensually, but responds to accommodation. * * * The myosis seems to be due to sympathetic paralysis. Tension same in both eyes. This unilateral reflex irido-plegia, which has been seen a few times, has in the cases reported been associated with mydriasis and not with myosis, as in this case. The left reflex iris paralysis indicates a lesion in the centrifugal portion of the reflex arc for the light reaction on the left side." (*Journal Nerv. and Ment. Dis.*, May, 1895.)

THE SO-CALLED BLEEDING POLYPUS OF THE NASAL SEPTUM.*

By W. Freudenthal, M. D.,
OF NEW YORK.

SINCE Victor Lange and Schadewaldt have drawn our attention to these bleeding tumors, they have been studied anew, especially by German observers. Several cases have been described in this country, but as a whole we must accept that their occurrence is quite rare.

The bleeding polypus is situated most frequently sesile at the anterior portion of the septum. We are often taken by surprise when we attempt to remove such an apparently plain polypus and encounter a tremendous hemorrhage. It is this long and sometimes dangerous bleeding which caused Schadewaldt to name them "bleeding polypi." But while this name is a very good one, from a clinical standpoint, yet it is otherwise objectionable, as "polypus" may mean histologically anything and everything. As a rule, these polypi present themselves histologically as angiomata in various forms.

The occurrence of these angiomata of the nasal septum is as mentioned, quite rare. Only recently (*Archiv. Ital. di Otol.*, 1895) there were collected 131 cases of tumors of the septum, of which ten were syphilitic, twenty-five tubercular, and sixteen not diagnosticated The remaining eighty tumors were: Sarcomata, twenty-nine; polypi, nineteen; carcinomata, eleven; papillomata, eight; angiomata, four; etc.

F. C. Cobb,[1] of Boston, collected nineteen cases of angiomata, but his own case is recorded as "a small growth

* Demonstrated before the German Medical Society of the City of New York, April 5, 1895.

[1] A case of angioma of the nasal septum. *Handbook of the First Pan-American Medical Congress*, Washington, 1893.

attached by a thin pedicle inserted into the septum." It occurred in a girl 15 years of age, anemic, who had suffered since six months from obstruction and bleeding from the right nostril. The growth was removed with "very slight hemorrhage," which is very rare in such cases. Besides the site healed completely in a few days, although the base had *not* been cauterized.

The case described by Victor Lange,[2] which is very interesting, corresponds more to the typical picture of these cases, and is as follows:

A woman, 29 years of age, suffered since five weeks from hemorrhages from the left nostril that were at times very severe. He saw a black, globe-like tumor which was seated with its broad base at the anterior portion of the septum, upon the slightest touch a profuse hemorrhage set in. After several unsuccessful trials he finally removed the tumor with a sharp curette.

He called it a soft fibroma, and says that these tumors must be rare, as among his many thousands of cases he only found six similar to the above mentioned. If we take the twelve cases reported by Schadewaldt, Alexander, Heymann and Scheier (*Archiv. für Laryngologie*, 1894, p. 259) and the three cases of Jonathan Wright, mentioned in his "Critical and Desultory Remarks, etc.," (*New York Medical Journal*, 1894, p. 364) we have all the cases reported up to date, and we must say that their number is surely very small, especially if we compare this with the thousands of "polypi" we see in other parts of the nose.

It is peculiar that the majority of these, if not all, have been noticed in women, but when Schadewaldt remarks that they might possibly have something to do with vicarious menstruation, as described long ago by B. Fränkel, I think he is greatly mistaken. The vicarious menstruation has quite another etiology, and as I have shown elsewhere,[3] they arise generally from other parts of the nose.

According to my opinion it is simply a traumatism that produces these growths, a traumatism that is directed against

[2] Ueber einen seltenen Fall von Septum polypen. *Wien. Med. Presse*, 1892, p. 2071.

[3] Severe hemorrhage from the nose. *The Post-Graduate*, March, 1894.

a place which naturally is supplied with an unusually large number of blood vessels. I imagine that if a blow or a strike or a fall is directed against this "Kiesselbach's" place in a manner so that it occurs as a direct blow, and no vessels are broken at once, this irritation causes in a very short time the formation of a new growth which, to a great extent, is composed of blood vessels.

I had never seen such a case until last winter when I had occasion to watch one during many months. Peculiar as it often is with the multiplicity of rare cases, a few weeks ago I saw another only much slighter in every respect. The history of the first case is briefly as follows:

Miss I. S., 22 years of age, a native of Russia, came to the German Poliklinik, complaining of nose bleeding, which had lasted for eight months. Since three months the bleeding was severer, without her having done anything for it. She attributes her trouble to a fall from a horse car, and says that soon afterwards she noticed that her right nostril was obstructed, and three months after the accident she could see a growth protruding from the nostril in looking into the mirror. On examination I could see, without a reflector, a round, reddish, bluish looking tumor of about 1 inch in diameter, seated at the anterior upper portion of the septum, covering, however, the locus Kiesselbachii. I must confess that at that moment I did not think of an angioma or the like, but tried at once to remove the polypus with a cold snare. But as soon as I touched the tumor such a severe hemorrhage set in that I hurriedly pulled out whatever I had in my loop. It was a small piece, but it took us half an hour before the profuse hemorrhage was stopped. I then sent her to St. Mark's Hospital, where I removed, with some difficulty, almost the entire tumor. Only a little stump was left. She grew so anemic from the loss of blood that in the next few days she fainted several times in the hospital, but she left it soon without having the stump cauterized. In the next few months I saw her but seldom at the Poliklinik. Now, *i. e.*, middle of March, she has terrible headaches before a bleeding occurs. At the same time the remaining stump gets larger. As soon as she bleeds, she feels easier. The bleeding occurs now three to four times daily, but not as much as before. She has besides tiring in the limbs, especially on the affected right side, and her general condition (great anemia, loss of flesh, lack of apetite) was so alarming that at this stage a fear for her life was not unfounded. She feared the hemorrhage, and for

that reason she did not want the cauterization done. At the same time her mental state was unsettled by the fear of the malignancy of her tumor. The reason for this fear was this: .

Two years ago I had seen her sister, Mrs. Z., whose history I might be allowed to mention here in parenthesis: — Mrs. Z., 32 years of age, was suffering for four months with pains in the right side of the throat, the cause of which was a *sarcoma of the tonsil.* Nine months before that she also complained of slight pains in the same part, but neither I nor one of my assistants, who thoroughly remembered the case, noticed at that time anything of a tumor of the tonsil. I advised the removal of the tumor per os. She was willing to have it done, but failed to appear on the fixed day. She had consulted somebody else, who sent her to the German Hospital for operation. By kindness of Dr. Kammerer I was present at the operation, which was performed by him by external incision after a preliminary tracheotomy. Although the operation was performed in a short time and very little blood lost, the patient died, like the other five similar cases which I had seen, of pneumonia (schluckpneumonie).

Now this girl constantly asked me whether she would not get the same tumor as her sister had. All the German writers agree that these tumors are perfectly benign, and the microscopical examination of her tumor also revealed, as we shall see later on, a *fibro-angioma.* Nevertheless, I hesitated in making an absolutely favorable prognosis, firstly on account of her sister's illness, and secondly on account of one case reported by Roe. This author saw a case that by microscopical examination proved to be an angioma, but it subsequently degenerated into an *angio-sarcoma.*

I finally convinced the girl that it was best to have the rest cauterized, and after this was done thoroughly, the bleeding stopped. I saw her six weeks later, and still see her from time to time, but in spite of good diet, iron, etc., her anemia is still great.

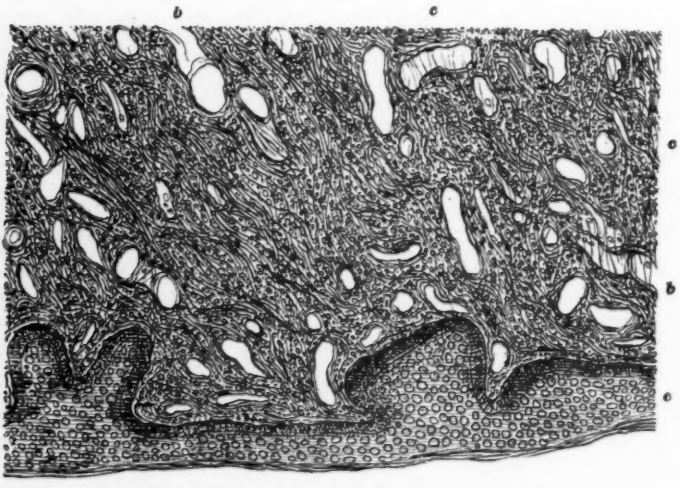
I will now give you the details of my microscopical examination:

The surface of the tumor is covered by a stratified epithelium. The breadth of the epithelial layer varies greatly according to blunt prolongations penetrating into the subjacent tissue. The layer of columnar epithelium everywhere sharply defined from the subjacent tissue. The latter exhibits irregular elevations reminding one of obtuse papillæ, to which, however, no elevations correspond on the surface of the tumor.

With the low power of the microscope the main mass of the tumor appears to be composed of a delicate fibrous connective tissue,

richly supplied with protoplasmic bodies. The latter mostly arranged in clusters, being least numerous in those portions of the tumor in which the fibrous structure is most pronounced. The tissue is everywhere traversed by numerous light fields, mostly empty, and only occasionally filled with red blood corpuscles. The smallest gaps are seen near the surface of the tumor, larger gaps of the type of arterial and venous blood vessels are found only in the central portions of the tumor. Besides, there are numerous light fields throughout the tissue, filled, apparently, with protoplasm, but not distinctly defined from the surrounding tissue. (See Fig. 1.)

FIG. 1.



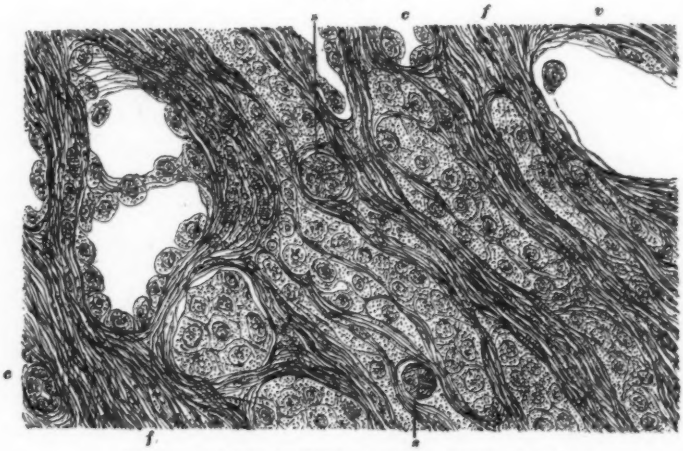
SECTION OF FIBRO-ANGIOMA OF NASAL SEPTUM. X 75.

e—Stratified epithelium. c c—Delicate fibrous connective tissue. b b—Blood vessels.

With the higher power of the microscope we recognize at the first glimpse bundles of fibrous connective tissue, varying in amount, though nowhere dense. Between these bundles we see vessels, mostly of the type of blood vessels, and again largely prevailing capillary blood vessels, whereas arteries and veins are comparatively scanty. Again the calibers of the capillary blood vessels vary very much. Some of the capillaries are narrow, slit-like, and straight; others are wider and more or less convoluted. The endothelial lining of all the blood vessels, especially of the capillaries, in many places conspicuous; in others it is torn away and clustered within the caliber of the vessel; or entirely absent, obviously by being

dragged away in the process of section cutting. The endothelia are without exception of large size. Although only a few vessels contain red blood corpuscles, I have no doubt the vast majority of the vessels of the tumor are blood vessels and not lymphatics. It is a well-known fact that newly formed blood vessels, such as we have before us, always are marked by the presence of large-sized endothelia. Besides I have nowhere seen a reticulum of coagulated fibrin within the calibers, such as we invariably meet with in lymph vessels. Special interest attaches to apparently solid functions made up of three or four endothelia. (See Fig. 2) There can be no doubt that we have before us stages of development of blood

FIG. 2.



SECTION OF FIBRO-ANGIOMA OF NASAL SEPTUM. X 300.

c c—Capillary blood vessels in transverse and oblique sections. s s—Solid formations of endothelia. f f—Loose fibrous connective tissue. v—Vein lacking endothelia.

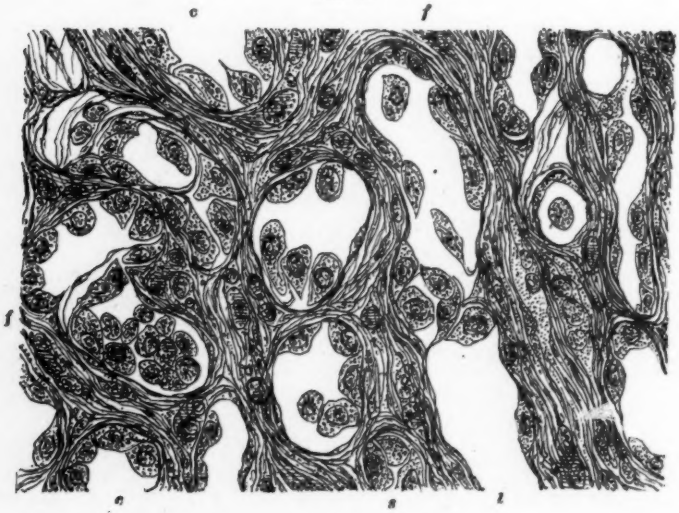
vessels, which latter, as is well established, are originally solid tracts of endothelia and afterward are hollowed out by a liquefaction of the central portions of the endothelia. (See Fig. 2.)

All doubts as to the nature of the vessels before us vanish upon carefully studying them with a magnifying power of 600 diameters. Again we observe large and distinctly nucleated endothelia either lining the walls of the capillaries or detached and clustered together owing to the process of section cutting. Exceptionally we meet with capillaries destitute of endothelia. The surrounding fibrous connective tissue is rather scanty wherever the blood vessels are

abundant. In this delicate fibrous connective tissue we see numerous protoplasmic bodies varying in size from that of nuclei to that of fully formed endothelia. This fact proves that the formation of capillaries in the tumor was progressing uninterruptedly. (See Fig. 3.)

There is no doubt that even a recurrence of the tumor may appear, although its nature must be considered entirely benign.

FIG. 3.



SECTION OF FIBRO-ANGIOMA OF NASAL SEPTUM. X 600.

c c—Capillary blood vessels lined by large endothelia in transverse section. l—Capillary blood vessel, with partly detached endothelia, in longitudinal section. s—Cluster of endothelia. f f—Delicate fibrous connective tissue.

The diagnosis from what has been said of the structure of the tumor is *fibro-angioma.*

The specimens of this tumor have been seen by Dr. C. Heitzmann, of this city, who corroborated the above diagnosis, and helped me greatly in finishing these drawings, for which I owe him thanks.

943 Madison Avenue.

ABSTRACTS OF PAPERS READ AT SEVENTEENTH ANNUAL CONGRESS OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION, ROCHESTER, N. Y., JUNE 17–19, 1895.

REPORTED BY M. D. LEDERMAN, M. D.,
OF NEW YORK.

Dr. J. W. Gleitsmann, of New York, read a very exhaustive and at the same time concise paper on "Surgical Treatment of Laryngeal Tuberculosis," in which he expressed his views upon the modern treatment of laryngeal phthisis. This paper must prove a valuable addition to the literature of the subject under consideration, as the author has collected all important references, dating from the initial surgical treatment of the disease, viz: tracheotomy for tubercular laryngitis in 1834. The first attempt at endo-laryngeal treatment was made by Mr. Marcet, as far back as 1869. He punctured the tubercular infiltrations, but his labors did not attract the attention of the profession at the time. It remained for Morris Schmidt to more fully dilate upon the treatment of this distressing affection, by incisions, which he did by publishing his method of operating in 1880.

Dr. Gleitsmann touches upon the various surgical methods suggested in the treatment of laryngeal tuberculosis: (1) Incisions; (2) submucoid injections; (3) galvano-cautery; (4) electrolysis; (5) curettment; and (6) extra laryngeal measures. Of these he considers curettment to be the most modern treatment, and strongly advocates its application in preference to all others. He has searched diligently for recorded opinions of those who have employed same, and out of seventy publications, he could only find six writers who spoke indifferently or unfavorably about it.

The author considers both sides of the question in a practical manner. He does not expect that the surgical treatment of the larynx "will directly exert a favorable influence on the almost always present pulmonary complications," but he states that we are justified in calling a patient cured of his laryngeal disease, "when, in spite of the continuance of the pulmonary disease, the laryngeal symptoms have subsided; when the larynx bears a normal aspect, and when no trace of the disease is found at a post mortem."

Up to the present time, no treatment has been tried which will prevent relapses, but Dr. G. thinks that, with proper curettment, relapses will be less frequent. Curettment is not suitable for all cases of laryngeal tuberculosis, and the more definite the indications are drawn, the better the results of the operation will prove to be. Some observers have claimed that curettment hastened the pulmonary process. The author does not deny the possibility of such a condition, but is inclined to believe that the aggrevated symptoms are merely a coincidence. Other objections brought forward are hemorrhage following the treatment, the painfulness and the difficulty of the operation. Bleeding can be controlled by a solution of lactic acid and perchlorid of iron. Strong solutions of cocain repeatedly applied will materially lessen the pain accompanying the surgical procedure. Practical manipulation will, no doubt, overcome the technical difficulty which was experienced by some operators.

In demonstrating the advantages of the remedy advocated, the speaker claimed that curettment is a rational proceeding, based upon sound surgical priciples. He drew the *simile* of a surgeon excising a tubercular joint, thus ridding the system of one source of infection, and remarks that the laryngeal operation acts in a similar manner. He agreed with the majority of operators, that this endo-laryngeal treatment, applied in properly selected cases, is by far better, quicker in action, and more effective, than any other method as yet suggested. The opinion that the operation was justifiable and should be performed before the disease has reached an inoperable stage, is strengthened by the knowledge that a patient is better able to battle against the ravages of this disasterous disease, if the ulcerative process could be limited to a single area.

Dysphagia is probably the most troublesome symptom of the local manifestation. This difficulty is caused by the infiltration of the arytenoid region, and in such cases the author strongly recommends curettment. Here the dense hard swelling can be removed

by Krause-Heryng's double curettes, generally at one sitting. The wound as a rule heals very rapidly, and the patient experiences great relief. It is for this reason that arytenoidectomy is in favor with most operators, in cases were curettment would otherwise be contra-indicated, viz: In active pulmonary disease with hectic. With the relief experienced, the individual is once again able to take sufficient nutriment, resulting in general improvement. The voice and cough is also benefited by surgical treatment, as cicatrization of the ulcerations takes place. Small, sharply defined ulcerations or dense localized infiltration, prove more amenable to treatment than extended shallow ulcerations or edematous structure. Bacteriological examination lends some weight to the prognosis.

Taking up the records of different operators, Dr. G. quotes the statements of Heryng, Gugenheim and Krauss, who, altogether, treated 455 patients. In some of these cases the larynx remained in healthy condition for one to eight years.

The author's experience is based upon twelve cases, all of whom had pulmonary complication. Some patients were operated upon for infiltration of the posterior laryngeal wall; in others the ventricular bands were excised, and arytenoidectomies were performed in some instances. "One patient died from heart failure, and another from advanced pulmonary disease. Four patients are without recurrence of laryngeal disease from six to ten months; one of whom had disease of the posterior laryngeal wall, another of the ventricular band, and two of the arytenoid region." The doctor mentions a case of tuberculous ulcer on the base of the tongue, first seen by him in 1888, and that during the summer the disease extended to the left tonsil, soft palate, the whole lingual surface of the epiglottis, and the left ary-epiglottic fold. Heroic treatment in this case was followed by a cure, the cicatricies of the palate and ary-epiglottic fold being plainly seen six years later. Referring to submucoid injections, Dr. G. calls attention to the initial work in this line by Krause and Heryng in 1886, and also to the suggestion of Dr. Major, who advised frequent and repeated injections at short intervals. The former investigators employed lactic acid as the medicament, while Dr. Chappell has derived excellent results from the use of an oily preparation of creasote.

Electrolysis and galvano-cautery are mentioned as being used by some operators, but the author prefers the more rapid and decisive method in the treatment of this lingering disease. Tracheotomy is favorably commented upon by a number of

authorities. Cases are cited in which the laryngeal disease materially improved after this operation was performed for serious stenosis. The author states that tracheotomy will always retain a prominent place in the treatment of laryngeal tuberculosis. The following indications for curettment are recommended:

"1. In cases of primary tuberculous affections without pulmonary complications.

"2. In cases with concommitant lung disease which is either in the incipient stage or has at least not progressed to softening and hectic conditions.

"3. It is best adapted for circumscribed ulcerations and infiltrations of the larynx especially.

"4. For the dense, hard swelling of the arytenoid region, the ventricular band, the posterior wall, for tubercular tumors and affections of the epiglottis.

"5. In advance lung disease with distressing dysphagia resulting from infiltration of the arytenoids, curettment is justifiable as the quickest means to give relief."

Contra-indications are:

"1. Advanced pulmonary disease and hectic.

"2. Disseminated tuberculous disease of the larynx, leaving little or no healthy tissue.

"3. Extensive infiltrations producing severe stenosis, when tracheatomy is indicated."

It is not to be recommended in nervous, distrustful patients, who lack the necessary perseverance or confidence in their physician.

IS ACUTE TONSILLITIS IN ANY WAY DEPENDENT UPON THE RHEUMATIC DIATHESIS?

Geo. B. Hope, M. D., of New York, read a paper in which he took the ground that the theory of acute tonsillitis very generally attributed to an underlying rheumatic or gouty diathesis is, in the writer's experience, not substantiated by clinical observation, and believes that the accepted version is largely due to the natural disposition to fall into line with time-honored views and unconsidered statements.

The issue is made that patients subject to attacks of tonsillitis do not commonly afford a history of rheumatism proximate or remote, while, on the other hand, the rheumatic individual rarely suffers from inflammation of the tonsil; that it is noteworthy that the tonsil in later life becomes less and less disposed to acute

attacks, while the rheumatic age is more confirmed. Furthermore, as a local acute manifestation rheumatism should select a sero-fibrous rather than a neuro-fibrous or lymphatic structure like the tonsil. Carrying the argument further, it is claimed that suppurative peri-tonsillitis is eminently of an infectious nature, and is frequently a sequela of intra-nasal operation quite independent of climatic or constitutional conditions.

The conclusion reached from the above is: that the favorite anti-rheumatic remedies as guaiac and the salicylates, as addressed to the causation are either erroneous in practice or act independently and by methods not clearly stated. Such remedies should not be considered specifics, but should be adjusted to the varying conditions of the subject. Moreover, it is not proven that the treatment does abort or mitigate the course of the disease.

TRANSLATIONS FROM CURRENT FOREIGN LARYNGOLOGICAL LITERATURE. (ABRIDGED).

By J. W. Gleitsmann, M. D.,
of New York.

A CASE OF CIRCUMSCRIBED GUMMATOUS ULCERATIVE TUMOR OF THE LARYNX.

Dr. E. W. Tschlenow of Moscow, (*Wiener Medicinische Wochenschrift*, 1895, p. 235), calls attention to the fact that in diseases of the larynx the seat of pathological lesion is of utmost importance in diagnosis. The thickening of the anterior surface of the posterior wall of the larynx without any other important symptoms suggests a suspicion of tuberculosis. Indeed sometimes one is entitled to make such a diagnosis. Although this is a very pathognomonic location for disease of the larynx, there are other seats very characteristic of the disease which, in addition to external appearance due to pathological changes, aid in diagnosis. I may refer to an article in Dr. J. Gottstein's book, 1893, page 316, which reads as follows:

"In diseases of the larynx, especially of the ventricular bands, the ary-epiglottic folds and the vocal cords, we notice a thickening which is diffused into the surrounding membrane, and, where the appearance of such lesion is not considerably changed, tubercular or syphilitic infiltration may be suspected." The same author also presents the following points for differential diagnosis between tubercular and syphilitic infiltration: "In tubercular thickening there is an even surface of a faint, pale and livid color. The syphilitic thickening is irregular, dark red in color, and is surrounded by a red inflammatory erea. The syphilitic infiltration ulcerates more rapidly than the tubercular and its process is very acute."

Prof. Schrötter confirms this last assertion by saying: "An excessively inflamed mucous membrane may, within twenty-four hours, be converted into a diffused ulceration." I now report the following case from Prof. Chiari's Klinik: J. M., 29 years of age, came to our Klinik on December 10, 1894, complaining of difficulty of swallowing, coughing at certain times, and also some hoarseness. He was in good health until nine years ago when he acquired a chancre with its usual complications. He was then referred to Prof. Auspitz. Five years after his first infection the patient began to complain of laryngeal symptoms, and was then referred to the laryngeal division of Prof. Schnitzler, where he was treated with iodoform insufflations, morphin internally, and later, with local applications of a 50% solution of lactic acid. This treatment was unavailing. He was now put upon the iodid of potassium treatment, and within three weeks he showed remarkable signs of convalescence. He was, however, only relieved for a short time as all his previous symptoms recurred, and being dissatisfied with this treatment he presented himself to the Poliklinik.

Examination of internal organs: At the right pulmonary apex there is slight dullness, yet at this time he seldom coughed, and expectorated very little. Arterio-sclerosis is found, but no other pathological lesion.

Laryngeal examination: The mucous membrane is slightly red, but almost normal, and at the upper portion of the anterior surface of the thickened arytenoid cartilage a pea-shaped tumor, the size of a hazelnut, can be seen. Its mucous membrane is smooth and is diffused into the surrounding membrane. Its color cannot be differentiated. On the upper surface of this tumor there is a slight contraction. The right side of the larynx is normal and both halves of the larynx move normally. The vocal cords are slightly reddened and do not approximate during phonation, leaving a spindle-shaped opening between the ligamentous portions. The cartilaginous part of the left cord cannot be seen, being covered by the tumor. The ventricular bands are normal. The trachea shows slight reddening.

After this examination the question of diagnosis arises; is it lues or tuberculosis? There is one more pathological lesion in this patient which assists us in making a positive diagnosis. The examination of his pharynx reveals an ulcerated swelling which covers the *pars oralis* and the *pars laryngea*. It extends upwards to the base of the uvula, downwards its border reaches the *aditus*

laryngis, and latterly to the palato-pharyngeal ligaments. The swelling is funnel-shaped and is covered with a fatty, dirty secretion. The gummatous swelling of the pharynx enables us to make a positive diagnosis of syphilis.

Treatment: In this patient the gummatous infiltration recurred frequently and disappeared readily under the potassium iodid treatment, eventually terminating very favorably. This treatment was discontinued and mercurial inunctions substituted. The ulceration of the pharynx was cauterized with nitrate of silver, an occasional spray of bichlorid of mercury 1 to 1,000 being used. The larynx itself was untreated. The examination of patient on March 14, 1895, revealed the cicatrix of the previous ulcerations upon the posterior wall of the pharynx. On the left arytenoid cartilage there appears a small red thickening, the remains of the swelling which was curetted two months previously. The patient received altogether twenty-five applications of unguentum hydrargyri amounting in all to 3 grams, and from the beginning of January until the end of February, 2 grams of potassium iodid were given daily.

ABSTRACTS FROM CURRENT RHINOLOGICAL AND LARYNGOLOGICAL LITERATURE.

By M. D. Lederman, M. D.,
of New York.

A CONSIDERATION OF THE VASCULAR MECHANISM OF THE NASAL MUCOUS MEMBRANE, ETC.

Dr. Jonathan Wright, Brooklyn, (*American Journal of the Medical Sciences*, Vol. CIX, No. 5). In an interesting paper the author describes the vascular anatomy of the turbinated bodies. Excellent illustrations accompany the writer's explanations. The venous sinuses, he states, depend largely for their expulsive power upon their muscular walls. In chronic inflammation of these parts the walls of the vascular spaces become much thickened by the overgrowth of non-elastic fibrous tissue. This interferes with the physiological action of the muscle fibers. In hypertrophic rhinitis there is a paresis of the walls as well as a dilatation of the lacunæ. The gradual absorption of this muscular tissue, due to the increasing pressure of the fibrous hyperplasia, accounts for the atrophic state. He further pictures the effect of the galvano-cautery upon the mucous membrane, and expresses the opinion that marked atrophy has resulted from its application.

TUBERCULAR TUMORS OF THE LARYNX.

Dr. J. P. Clark, Boston, Mass., (*Ibid*). After noting similar cases reported by other observers, the author details the history of his patient. The growth was found on the left venticular band in a woman 31 years of age. No tubercular history was elicited. It was a simple senile growth, covered with mucous membrane, concealing, during respiration, most of the left vocal cord and part of the right. The larynx was otherwise normal except for

a slight infection of the cords. Under cocain anesthesia the tumor was removed by means of the cold wire snare. Hemorrhage was very slight. The gross appearance of the growth resembled a fibroma. Examination of the lungs gave negative results. Microscopical examination of the tumor showed it to be composed of miliary tubercles. No bacilli were discovered.

HEMORRHAGIC PHARYNGITIS.

Dr. Nahier (*Ibid*). Effusions of blood from the pharynx occurred in a shopman, 29 years of age. These symptoms occurred twice; the quantity equaling a tumblerful. No lesion existed. The bleeding was controlled by an application of silver nitrate solution 1 to 8, followed by applications of citron-juice as home treatment.

ZINC STERATE IN THE TREATMENT OF ATROPHIC RHINITIS.

Dr. J. F. Gibbs (*The Canada Lancet*, Vol. 27, No. 5). The plan of treatment consisted in cleansing the nose by antiseptic sprays and peroxid of hydrogen. After the crusts were thoroughly removed the membranes were covered with a thin layer of powdered sterate of zinc containing 25% of europhen. Very satisfactory results were obtained.

(The stimulating action of the medicament employed was no doubt due to the europhen, as this drug contains about 20% or more of iodin as one of its constituents. M. D. L.)

REMOVAL OF THE TONSILS BY THE WIRE SNARE.

Dr. Marcel, Bucharest, (*Indian Medical Gazette*, Vol. 30, No. 5). The author's conclusions are based on a series of fifty cases operated upon by the above method. He claims that the cold snare is (*a*) less likely to provoke fright in nervous children; (*b*) the instrument is less costly; (*c*) can be more readily cleansed, and is rendered non-aseptic, especially if a new wire is used for every operation; (*d*) the removal of the tonsil is more thoroughly effected.

(We strongly advocate the employment of the Mathieu tonsillotome as a better instrument, and one which will accomplish the desired result in a neater manner and in much less time than the snare requires. M. D. L.)

DISEASE OF THE MIDDLE TURBINATED WITH PUS IN THE ETHMOID CELLS.

Dr. George R. McDonagh (*The Canadian Practitioner*, Vol. 20, No. 5). In the experience of this observer the disease usually resulted from trauma or from extension from the nasal cavities. Granulations were frequently found with thickening of the anterior portions of the middle turbinated body. The probe revealed dead bone. Polypi formed from irritation of the mucous membrane. At the seat of granulation the bone may be found cleft with pus exuding. Headache, tightness over the bridge of the nose with neuralgia are some of the symptoms existing. Trans-illumination is an aid to the diagnosis. The inner half of the bone should be removed, and the ethmoid cavity cleansed with hydrozone or iodoform and glycerin.

COCAIN IN CHLOROFORM NARCOSIS.

Dr. Rosenberg (*Ibid*). At a meeting of the Berlin Medical Society the author advised the spraying of the nasal mucous membrane with a cocain solution (per cent not mentioned) before administering the chloroform. He states that by this treatment anesthesia is more rapidly effected, and reflex action on the heart is prevented. Cocain being an antidote to chloroform its absorption would probably lessen the danger of the latter.

ECCHONDROMA ARISING FROM NASAL BONE.

Dr. H. C. Barclay, Waimate, (*New Zealand Medical Journal*, Vol. 8, No. 1). The growth originated from the right nasal bone. It was noticed four years before the reporter saw his patient. History of increasing swelling. The young man was 27 years of age, and dated his trouble from knocks received while playing football. The tumor spread from the anterior to the posterior nares. Half of the nasal bone was involved in the growth, so same was removed with a bone forceps after an external incision was made. Perfect union resulted.

A CASE OF CONGENITAL ATRESIA OF THE NASO-PHARYNX.

Dr. C. B. Storrs (*Ontario Medical Journal*, Vol. III, No. 9). This anomaly was discovered in a female 7 months old. Mother noticed difficulty in breathing with loud wheezing and rattling.

On examining the anterior nares the author found the nostrils to be imperforate, the membrane extending entirely across them at the level of the inferior turbinated bone. No probe could be passed. Under anesthesia an incision was made, and the opening dilated with long dressing forceps. It was necessary to repeat the dilatation as the opening showed a tendency to close.

SADDLE-BACK NOSE.

Dr. L. A. Stimson (*Annals of Surgery*, Vol. 21, No. 6). In the case reported the deformity resulted from a broken nose. A canoe-shaped piece of aluminum five-eighths of an inch long was inserted between the skin and bones through a small incision on the ala, thus raising the bridge of the nose to its proper line. The wound healed nicely, but the outline of the nose was not exactly straight. The metal splint was replaced by a similar one of gutta percha about half as large again. The desired effect resulted.

(If these splints can be worn without exciting inflammatory changes the procedure is a valuable addition to nasal surgery. There is a wide field for cosmetic operations in this locality. M. D. L.)

PROFESSIONAL NEWS.

Dr. Casey A. Wood of our Editorial Staff has gone to Europe for a three months' vacation. Dr. Wood will visit a number of foreign Kliniks and attend the meeting of the Heidelberg Ophthalmological Society. When his wanderings are over we hope to profit by his added experiences abroad.

MARRIED.—We are pleased to announce the recent marriage of Dr. M. A. Goldstein of our staff of editorial collaborators to Miss Leonore Weiner, both of St. Louis. We extend our congratulations.

BOOK NOTICES.

SKIASCOPY AND ITS PRACTICAL APPLICATION TO THE STUDY OF REFRACTION. By Edward Jackson, A. M., M. D., Professor of Diseases of the Eye in the Philadelphia Polyclinic, Surgeon to Wills Eye Hospital, etc., 8 Vo., 112 pages with twenty-six illustrations. Price $1.00. The Edwards & Docker Co., Philadelphia.

Considering the wide practical value of the shadow-test it seems strange that this book or something of the kind has not been published before. As one method of objectively measuring refraction, some practical acquaintance with skiascopy is essential to every working ophthalmologist; and if as the author states, with a few days' practice the merest tyro may be able by it to estimate the refraction in favorable eyes with an accuracy not to be attained by any other objective method, it will certainly claim some attention from the "general practitioner" who wishes to know when a patient has an error of refraction that should be corrected.

It must be noted, however, that Dr. Jackson does not teach that skiascopy is to be mastered with a few days' practice. Of the first chapter a considerable portion is devoted to the difficulties of the test and suggestions as to how to study it. The remainder of this chapter is occupied with an account of its multitudinous names and its history. Succeeding chapters are given to General Optical Principles, Conditions of Accuracy, Regular Astigmatism, Aberration and Irregular Astigmatism, Practical Application with the Plane Mirror, Practical Application with the Concave Mirror, and General Considerations including Apparatus, Mydriatics and the Relative Advantages of the Plane and Concave Mirrors.

The work of the printers has been well done, type of good size, good paper, and substantial binding, all add to the appearance of the volume. The illustrations are nearly all original, the majority of them showing the appearance of light and shadow in the pupil in different conditions of refraction.

WILLS EYE HOSPITAL REPORTS, Vol. I., No. 1, January, 1895. Published by the Editorial Committee of Wills Eye Hospital, Philadelphia.

That Wills Hospital, with its 13,000 eye patients a year, furnishes the clinical material upon which can be based an extremely valuable series of hospital reports there can be no doubt, and the papers in the first number give the best indication that such a series is forthcoming. We find no indication of the intervals at which these parts are to appear; but considering that the one before us includes 136 octavo pages, with 25 illustrations, some of them colored, it seems hardly likely that they will be issued oftener than once in six months or a year.

Among the articles of special interest to the ophthalmologist we note a report of three cases of Cilia in the Anterior Chamber, by Dr. Geo. C. Harlan; one on Blepharoplastics, by Dr. P. D. Keyser; Clinical Methods and Memoranda, by Dr. H. E. Goodman; Acute Glaucoma from Atropia in a Child Twelve Years Old, by Dr. Frank Fisher; Clinical Cases, by Dr. C. A. Oliver; a case of Corneal Opacity Following the Use of a Lead Lotion, by Dr. S. D. Risley; and on the Limitation, Subsidence, and Disappearance of Opacity after Injury of the Crystalline Lens, by Drs. Edward Jackson and T. B. Schneideman.

Dr. Conrad Berens describes some new Blanks for Sketching the Ocular Fundus; Dr. Schwenk writes on Pediculiciliaris, and reports twenty cases; Drs. Zentmayer and Posey discuss the Comparative Value of Eserin and Iridectomy in Simple Glaucoma, basing their conclusions on 167 cases; and not the least in value are the reports of the Resident Physicians, Drs. Ellett, Parker and Curry, on the Cataract Operations and cases of Serious Injury of the Eyeball. An historical sketch of the institution by Dr. Berens, and a list of its medical and surgical officers from the time of its organization in 1833 to the present are included.

Of course the time has gone by when hospital reports, even the most valuable, could be the main dependence of the working ophthalmologist to keep him abreast of the progress of his specialty, but such as the one before us will prove a very valuable addition to every ophthalmic library.

A System of Legal Medicine. By Allen McLane Hamilton, M. D., Consulting Physician to the Insane Asylums of New York, etc., and Lawrence Godkin, Esq., of the New York Bar, with the collaboration of thirty of the most eminent medical and legal writers of the last years of the Nineteenth Century. To be completed in two volumes. Price, cloth, $5.50 per volume; sheep, $6.50 per volume. Published and for sale by E. B. Treat, 5 Cooper Union, New York, N. Y.

The first volume of this "System of Legal Medicine" is before us. It contains 657 pages. The first chapter is a scholarly introduction by Lawrence Godkin, Esq., in which definitions of legal

medicine and experts are given, followed by a review of the progress in legal medicine, etc. The other fourteen chapters are by learned collaborators, and are as follows:

1. Medico-Legal Inspections and Post-Mortem Examinations. By Algernon T. Bristow, M. D.

2. Death in its Medico-Legal Aspects. By Francis A. Harris, M. D.

3. Blood and other Stains—Hair. By Prof. James F. Babcock.

4. Identity of the Living. By Allan McLane Hamilton, M. D.

5. Identity and Survivorship. By Benjamin N. Cardozo, Esq.

6. Homicide and Wounds. By Lewis Balch, M. D.

7. Poisoning by Inorganic Substances. By Charles E. Pellew, Ph. D.

8. Poisoning by Alkaloids and Organic Substances. By Walter S. Haines, M. D.

9. The Toxicologic Importance of Ptomaines and other Putrefactive Products. By Victor C. Vaughan, M. D.

10. The Medical Jurisprudence of Life Insurance. By Brandreth Symonds, M. D.

11. Accident Insurance. By Cortlandt Field Bishop, Esq.

12. The Obligations of the Insured and the Insurer. By R. C. McMurtrie, Esq.

13. Of Certain Legal Relations of Physicians and Surgeons to their Patients and One Another. By William A. Purrington, Esq.

14. Indecent Assault Upon Children. By W. Travis Gibb, M. D.

Every chapter in this volume is well written, showing that each writer has a thorough understanding of his subject. The chapter on "Toxicologic Importance of Ptomaines and Other Putrefactive Products" by Victor C. Vaughan, M. D., is not as complete as the readers of the ANNALS might expect, as it is a very important subject, but the author has exercised great care in presenting the subject in a conservative and guarded manner, leaving nothing unexplained. This short chapter of eighteen pages, alone, is worth the price of the book to any well-informed physician, though every chapter will be helpful to the medical man who is likely to be called before courts of justice in these days when the people appear to be made for the courts, instead of the courts for the people.

This "System of Legal Medicine" should be in the library of every physician as the legal rules and forms elucidating questions relating to the cause or time of death, conception and birth, or the cause or effect upon the legal status of individuals of mental or physical disease or injuries are such that the physician will profit by reading them though he may never be so unfortunate as to be called into court as an expert. In these days of criticism and belittling of expert testimony no member of the medical profession

can afford to not acquire a thorough knowledge of all that relates to medical jurisprudence, as it is his learning and experience, drawn from the "myriad of single instances," which qualify the medical expert and gives his opinion in the specific instance in issue, gravity and weight.

THE YEAR-BOOK OF TREATMENT FOR 1895. A Comprehensive and Critical Review for Practitioners of Medicine and Surgery. In one 12mo volume of 501 pages. Cloth, $1.50. Philadelphia: Lea Brothers & Co., 1895.

This issue of the *Year-Book* is equal, if not superior, to previous issues.

Its contents show that it is not intended to include everything new, but only a share of that which promises to be of permanent value to the therapeutist. The reviewer cannot help thinking that the value of the *Year-Book of Treatment* would be greatly enhanced by having it edited by an American, *e. g.*, Roberts Bartholow. Our English brethren do not appear to realize that such a book should take in more "territory" in order to make the *Year-Book* useful to all English reading physicians.

Contracted ideas retard the progress of rational therapeutics. The name of the editor does not appear, but the work has been done by twenty-three well-known English specialists.